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# THE INTERPRETATION OF THE VI-TESTS*

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In South Africa the Vi-agglutination test is widely used in attempts to trace chronic carriers of *Salmonella typhi*. Although it is often deplored that *S. typhi* is seldom isolated from the Vi-reactors, some 20,000 tests are requested each year of this Institute.[1] To determine the significance of a positive Vi-reaction, it was decided to investigate a number of sera by the Vi-test and check the reactors a second and a third time. Individuals who showed Vi-agglutinins on 3 occasions were to be considered definite reactors, and their excreta were to be subjected to repeated bacteriological examinations (50 if necessary) to detect carriers. The plan broke down twice owing to circumstances beyond our control.

Of 465 persons examined for Vi-agglutinins, 410 (88·2%) were negative in the first dilution of 1:10; only 8 (1·7%) were positive on 3 or more occasions, but their titres fluctuated between 1:40 and less than 1:10. Three of them refused further cooperation, whereas the remaining 5 had their excreta examined from 2 to 31 times; *S. typhi* was not isolated. These disappointing results once more raise the question of the value of this test. Equally important, but often conveniently ignored, is the problem of the frequency with which a positive Vi-reaction casts suspicion on a non-carrier.

Since the discovery of the Vi-antigen by Felix and Pitt,[2] the tube agglutination technique for demonstration of Vi-agglutinins has remained fundamentally unchanged, although individual workers have introduced certain modifications, particularly in antigen preparations and incubation periods. As a modification of the original technique, Saint-Martin and Desranleau[3] devised a rapid micro-method, and Spaun[4] converted the test into a haemagglutination reaction by coating human erythrocytes with dissolved Vi-antigen.

On the value of the tests for tracing chronic typhoid carriers, opinions differ. Some authors[5,6] agree with Felix's statement[7] that it is 'an aid to the detection of chronic typhoid carriers', but acknowledge the occurrence of Vi-agglutinins in healthy individuals, and their absence from a certain proportion of carriers.

A second group of authors is more enthusiastic. Saint-Martin and Desranleau[3] think that it is an efficient screening test. Pijper and Crocker[8] regard all Vi-reactors as carriers. Rische and Rohne[9] suggest that all individuals with a Vi-titre of 1:20 or more should be prevented from handling food, and Lewin,[10] discussing the application of the Vi-test, says: 'Obviously, serological tests (Vi) must be continued as a routine measure; by preventing all positive Vi-reactors from handling milk and foodstuffs many typhoid carriers would be eliminated, but it must be realized that many "normal" reactors would be penalized'.

The third group doubts its value. Davis[11] does not deny that some of his sera with low Vi-titres were possibly derived from carriers, but he thought that they were frequently of no significance. Radowsky[12] agreed that low titres did not necessarily indicate the existence of the carrier state. Spaun[13] considered the haemagglutination test unsuitable for the detection of chronic carriers, since at least 12% would be missed. Joe *et al.*,[14] in Indonesia, found that 14·7% of 'normal' people were Vi-reactors and concluded that as a screening test it was unreliable. In the experience of Manson-Bahr,[15] the Vi-test was of little practical value in areas with a high endemicity of typhoid.

Thus, the usefulness of the Vi-test in detecting chronic typhoid carriers is not universally accepted and deserves close examination.

## PUBLISHED METHODS

From the abundant literature on the Vi-test those publications have been selected in which the test has been carried out quantitatively on clearly defined groups. The carriers must be bacteriologically confirmed; the controls shown to be free of *S. typhi*. Eleven papers fulfilled these criteria, but the techniques varied.

Felix[7,16] used the Watson strain, alive or formalinized, incubating 2 hours at 37°C followed by 24 hours at room temperature. Standard serum was incorporated as a control.

Horgan and Drysdale[17] used the same technique, except that the living antigen suspension was prepared from the Bhatnagar strain, Vi I.

Klein[18] incubated the tests for 24 hours at 37°C, but otherwise followed the method described by Horgan and Drysdale.[17]

Lewin, Bersohn and Hogg[5] prepared a formalinized antigen suspension from strain Vi I Bhatnagar. The tubes were incubated for 2 hours at 37°C and then centrifuged at 3,000 r.p.m. for 2 minutes. Standard serum was incorporated.

Ruhnke[6] prepared his, apparently living, suspension from strain 965 (Rauss). The incubation period was 3-4 hours at 37°C followed by 18 hours at room temperature. Standard serum was not mentioned.

Rische and Rohne[9] used strain 58907 (Robert Koch Institute, Berlin). The sera were inactivated and incubated with a presumably living suspension for 3 hours at 37°C. Standard serum was not mentioned.

Ortel[19] worked with a living suspension of strain 965 (Rauss). The sera were inactivated. He incubated for 1 hour at room temperature, followed by 2 hours at 37°C. Standard serum was not mentioned.

* Read in abbreviated form at the 41st South African Medical Congress (M.A.S.A.), Durban, September 1957.

Saint-Martin and Desranleau[3] used a glycerolated antigen prepared from the Bhatnagar, Vi I. In contrast to the above-mentioned authors, they employed a slide technique, in which the slide was agitated for 4 minutes and then left for 30 minutes at room temperature. The test was read under a stereoscopic microscope at a magnification of 72. Standard serum was not mentioned.

Staack and Spaun[20] coated erythrocytes of human type O with a Vi-antigen extract of strain Vi I.[4] The treated erythrocytes were used in a haemagglutination test which was incubated with inactivated serum for 1 hour at 37°C. Standard serum was not mentioned.

Landy and Lamb[21] used a similar haemagglutination technique, with a purified Vi-antigen prepared from *E. coli* 5396/38, acetone-killed and dried. The test was incubated at 37°C for 2 hours. Standard serum was incorporated.

### PUBLISHED RESULTS

Table I records some authors' Vi-results among chronic carriers of *S. typhi*. Using the tube agglutination method, Klein[18] and Rische and Rohne[9] found approximately 10% of the sera were negative in the lowest dilution, 1:5. At a 1% probability level (Fisher's exact probability test) their results do not differ from those of Felix[7] and Ortel.[19] By contrast, there is a significant difference between the percentage of sero-negative typhoid carriers observed by Felix[7] (0%) and by Ortel[19] (33%). This is not due entirely to a difference in antigenic sensitivity, since several authors incorporated standard sera, and yet arrived at different results. Several other variables influence the reaction and to achieve a comprehensive picture of the kinetics, each one should be evaluated separately.

TABLE I. VI-AGGLUTININS IN PROVEN TYPHOID CARRIERS

| | *Tube Agglutination* | | | | | | *Slide Aggl.* | *Haemaggl.* | |
|---|---|---|---|---|---|---|---|---|---|
| *Author Year* | *I '38* | *II '43* | *III '45* | *IV '50* | *V '51* | *VI '52* | *VII '51* | *VIII '53* | *IX '53* |
| No. of sera | 56 | 41 | 4 | 71 | 63 | 24 | 26 | 58 | 20 |
| Neg. react. in lowest dil. | 0 | 4 | 2 | 11 | 7 | 8 | 0 | 7 | 2 |
| End titre of pos. sera: | | | | | | | | | |
| 1:2 .. | — | — | — | — | — | — | 4 | — | — |
| 1:5 .. | 18 | 2 | 0 | 9 | 0 | 0 | } 5 | 4 | 2 |
| 1:10 .. | 18 | 12 | 0 | 12 | 4 | 2 | | 12 | 2 |
| 1:20 .. | 11 | 13 | 0 | 19 | 18 | 5 | | 16 | 4 |
| 1:40 .. | 4 | 6 | 0 | 11 | 23 | 5 | } 17 | 9 | 5 |
| 1:80 .. | 3 | 3 | 1 | 9 | 9 | 3 | | 3 | 4 |
| 1:160 .. | 1 | 1 | 1 | | 1 | 0 | | 5 | 1 |
| 1:320 .. | 1 | | | | 1 | 1 | | 2 | |

I. Felix.[7] II. Klein.[18] III. Lewin, Bersohn and Hogg.[5] IV. Ruhnke.[6] V. Rische and Rohne.[9] VI. Ortel.[19] VII. Saint-Martin and Desranleau.[3] VIII. Staack and Spaun.[20] IX. Landy and Lamb.[21] — = not done

Notwithstanding certain justifiable objections to the pooling of the results, the general trend may be reflected by combining the first 6 columns of Table I (Table II). It is noteworthy that an average of 12·4% of the typhoid carriers are Vi-negative in the 1:5 dilution. If a positive reaction at a serum dilution of 1:10 is accepted as the lowest diagnostic titre—and this is often done—then 23·6% or almost one-quarter of the carriers will go undetected. Thus, if the test is to be of practical value, a positive reaction of 1:5 must, following Felix,[7] be accepted as diagnostic but, in spite of this, 10 - 15% of the carriers will have eluded detection by the Vi-agglutination test.

TABLE II. VI-TITRES AMONG 259 PROVEN CHRONIC TYPHOID CARRIERS (COMBINED RESULTS)

| | *Final ser. dil.* | *Number* | *Per cent.* |
|---|---|---|---|
| Negative | 1:5 | 32 | 12·4 |
| Positive | 1:5 | 29 | 11·2 |
| ,, | 1:10 | 48 | 18·5 |
| ,, | 1:20 | 66 | 25·5 |
| ,, | 1:40 | 49 | 18·9 |
| ,, | 1:80 | 28 | 10·8 |
| ,, | 1:160 | 4 | 1·5 |
| ,, | 1:320 | 3 | 1·2 |

The results in Table III show that sera from a number of apparently healthy people give a positive Vi-reaction. On the whole the titres are lower than those found in typhoid carriers. The proportion of 'false positive Vi-reactions' varies between 46·5% and 3·2%[17,19]. The differences are significant at the 1% probability level. In keeping with the previous arguments we may also combine the results of Table III (Table IV). It is seen that of 1,589 sera, 181 (11·4%) were positive, and that the proportion of those giving false positive reactions decreases rapidly with increasing titre. In this control material there is not a single sero-reactor of 1:80 or higher.

TABLE III. VI-AGGLUTININS IN ALLEGED NON-TYPHOID CARRIERS

| | *Tube Agglutination* | | | | *Slide Aggl.* | *Haem-aggl.* |
|---|---|---|---|---|---|---|
| *Author Year* | *X '40* | *III '45* | *V '51* | *VI '52* | *VII '51* | *VIII '53* |
| No. of sera .. | 200 | 103 | 204 | 1,082 | 9,192 | 243 |
| Neg. react. in lowest dil. .. | 107 | 87 | 167 | 1,047 | 9,082 | 225 |
| End titre of pos. sera: | | | | | | |
| 1:2 .. | — | — | — | — | 59 | — |
| 1:5 .. | 55 | 8 | 15 | 14 | } 22 | |
| 1:10 .. | 37 | 2 | 15 | 14 | | |
| 1:20 .. | 1 | 6 | 2 | 5 | 29 | } 18 |
| 1:40 .. | | | 5 | 2 | | |

III. Lewin, Bersohn and Hogg.[5] V. Rische and Rohne.[9] VI. Ortel.[19] VII. Saint-Martin and Desranleau.[3] VIII. Staack and Spaun.[20] X. Horgan and Drysdale.[17] — = not done.

TABLE IV. VI-TITRES OF 1,589 APPARENTLY HEALTHY INDIVIDUALS (COMBINED RESULTS)

| | *Final ser. dil.* | *Number* | | *Per cent* | |
|---|---|---|---|---|---|
| Negative | 1:5 | 1,408 | | 88·6 | |
| Positive | 1:5 | 92 | } 181 | 5·8 | } 11·4% |
| ,, | 1:10 | 68 | | 4·3 | |
| ,, | 1:20 | 14 | | 0·9 | |
| ,, | 1:40 | 7 | | 0·4 | |

The percentages given in Tables II and IV may be rearranged so that the cumulative total at each serum dilution is obtained (Table V). There is a higher percentage of

TABLE V. CUMULATIVE PERCENTAGES OF VI-REACTORS AT VARIOUS SERUM DILUTIONS AMONG TYPHOID CARRIERS AND CONTROLS

| | *Final ser. dil.* | *Typhoid carriers* | *Controls* |
|---|---|---|---|
| Negative | 1 : 5 | 12·4% | 88·6% |
| Positive | 1 : 5 | 87·6% | 11·4% |
| ,, | 1 : 10 | 76·4% | 5·6% |
| ,, | 1 : 20 | 57·9% | 1·3% |
| ,, | 1 : 40 | 32·4% | 0·4% |
| ,, | 1 : 80 | 13·5% | |
| ,, | 1 : 160 | 2·7% | |
| ,, | 1 : 320 | 1·2% | |

Vi-reactors among typhoid carriers than among the non-carrier group. Moreover, the Vi-reaction becomes increasingly specific as the titre rises. For example it appears to be almost specific when the titre is 160, but at that level 97% of the carriers are non-reactive. On the other hand, if the sensitivity is to be increased so that the reaction detects 87% of the carriers (1:5 dilution) one has to forfeit specificity. At this serum dilution, 11·4% 'false positive reactors' would occur among the general population, which is similar to the findings elsewhere.[14,22] In most centres relying upon the Vi-test in tracing typhoid carriers, it is usual to compromise and accept a Vi-titre of 1:10 or 1:20 as diagnostic. If 1:10 is accepted, one-quarter of the carriers would be missed and 5·6% of the non-carriers would be positive; if 1:20 is considered diagnostic, almost half of the carriers would be considered free of typhoid and 1·3% of the non-carriers would give false positive results.

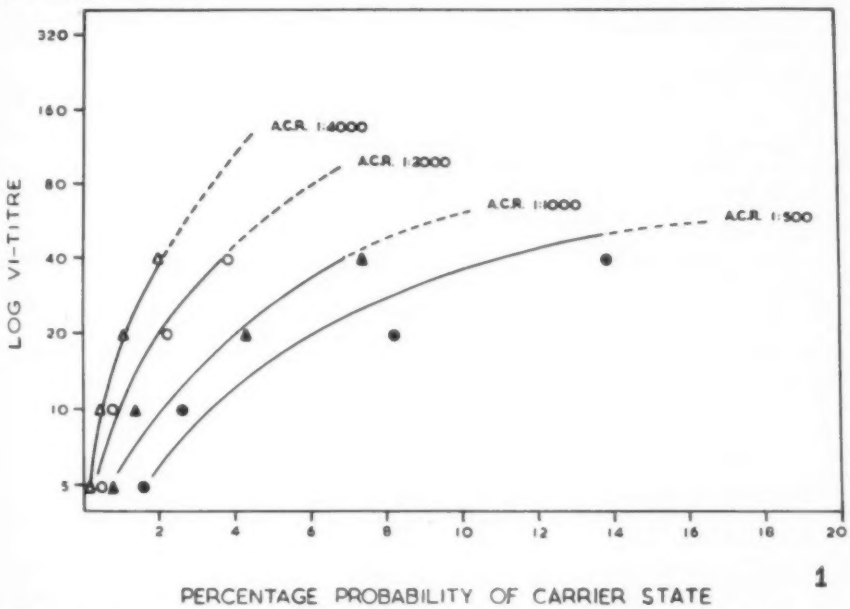


Fig. 1 Diagnostic value of Vi-agglutination titres in populations with various carrier rates. (A.C.R. = assumed carrier rate.)

In a given population the percentage probability that a Vi-positive result signifies a typhoid carrier may be expressed as:

$$\frac{(\text{No. of Vi-positive carriers in the population}) \times 100}{\text{Total no. of Vi-reactors in the same population}}$$

Ames and Robins[23] calculated statistically that there were 2,490 typhoid carriers in New York in 1940, or 1 carrier to 2,500 non-carriers, of whom only 17% were known to the health authorities. They quoted the carrier rate in Missouri as 1:3,500, but pointed out that these were minimum figures. Assuming that the carrier rate is considerably higher, e.g. 1:1,000, then there would be 100 carriers in a population of 100,000. At a 1:5 level the carriers would provide 88 Vi-reactors (Table V), whereas the non-carriers at the same level would produce 11,389 reactors. The probability of any one of the reactors being a carrier would then be $88 \times 100 \div 11{,}477 = 0{\cdot}8\%$. The probability varies with the actual carrier rate,

TABLE VI. THE PROBABILITY OF DETECTING TYPHOID CARRIERS BY MEANS OF THE VI-REACTION

| *Vi-titres* | *Assumed carrier rates* | | | |
|---|---|---|---|---|
| | 1 : 500 | 1 : 1000 | 1 : 2000 | 1 : 4000 |
| 1 : 5 | 1·5% | 0·8% | 0·4% | 0·2% |
| 1 : 10 | 2·6% | 1·3% | 0·7% | 0·3% |
| 1 : 20 | 8·2% | 4·3% | 2·2% | 1·1% |
| 1 : 40 | 14·0% | 7·5% | 3·9% | 2·0% |

the Vi-titre, and the proportion of 'false positive' Vi-reactors in the population. The first two factors are considered in Table VI, the last being regarded as a constant. By plotting the values on semi-logarithmic paper (Fig. 1) we obtain a family of graphs. The positions of the first points on the graphs support the existence of curves rather than straight lines. The relatively small number of high-titred Vi-sera obscures the continuation of the graphs, but it is possible that they will asymptotically approach the parallels to the abscissa.

It is evident from these graphs that if the carrier rate is 1 : 1000 and the criterion chosen is a serum titre of 1 : 10, then there is a 1·4% probability that such a serum will originate from a typhoid carrier, but 98·6% likelihood that it is drawn from a non-carrier. On the other hand, with a carrier rate of 1 : 4000 the probability of detecting a carrier from sera of the same titre (1 : 10), would be 0·3%, that is to say, more than 300 normals would be included for every carrier so detected. Increasing the diagnostic titre to 1 : 40 would again narrow

the field to 50 normals per carrier, while still excluding two-thirds of all carriers. These examples illustrate that the diagnostic value of a given Vi-titre varies proportionately with the prevalence of typhoid carriers.

The probability of a titre being evidence of the carrier state is given in Table VII, which is based on an assumed carrier rate of 1:1,000, but may, of course, be adapted to suit any other rate.

TABLE VII. THE SIGNIFICANCE OF INCREASING VI-TITRES (ASSUMED CARRIER RATE 1 : 1000)

| *Vi-titre* | *Probability of Vi-reactors being carriers* | *Probability of Vi-reactors being non-carriers* | *Probability of missing carriers (100 − sensitivity %)* |
|---|---|---|---|
| 1 : 5 | 0·8% | 99·2% | 12·4% |
| 1 : 10 | 1·4% | 98·6% | 23·6% |
| 1 : 20 | 4·3% | 95·7% | 42·1% |
| 1 : 40 | 7·4% | 92·6% | 67·6% |

A satisfactory compromise for a diagnostic titre appears unattainable. The test may yet have an application in the search for a carrier known to be present in a closed community, since it may narrow the field of cultural examination. Should the carrier not be found among the Vi-reactors, it must be remembered that Vi-negative carriers occur in a percentage which increases with the stringency of the titre employed.

The conclusion to be drawn is that it is unrewarding to search at random for typhoid carriers by the Vi tube agglutination technique; on the other hand, if it is reasonable to assume that there is a carrier in a small group of 10 - 50 individuals, the test may be of some assistance, particularly if its limitations are realized.

The question arises whether a better result can be expected from slide or haemagglutination tests. Saint-Martin and Desranleau[3] found that all typhoid carriers gave a positive slide agglutination reaction with their Vi-antigen, though 16% were very weak (Table I). The specificity was 98·8% (Table III). Assuming a carrier rate of 1:1000, this gives an 8·3% probability of a Vi-reactor in the lowest dilution being a carrier and, of course, increased probabilities with higher titres. Theoretically, this test appears to be more satisfactory than tube agglutination, but other workers like Richter[24] and Rische and Rohne[9] have been unable to confirm the advantages of the slide agglutination.

The haemagglutination test[20,21] has practically the same sensitivity and specificity as the tube agglutination test. Accordingly, in its present form it offers no advantage over the classical method.

It should be pointed out that sensitivity, specificity and probability are not the only factors interfering with the interpretation of the Vi-results; for example, the degree of reproducibility as well as the influence of prophylactic immunization are relevant factors, but the assessment of these is beyond the scope of this paper.

The findings and arguments in this paper are the results of combining the observations made by a number of individual workers. The pooling, it may be argued, would be unjustified if all the tests were unsatisfactory, or if one or more tests were superior to the others. In the former case further discussion would be futile, while in the latter, the better methods should be promoted and the unsatisfactory tests abolished. The figures set out in Tables I and III do not indicate the superiority of any particular modification.

## CONCLUSION AND SUMMARY

From the literature it appears that a wide range of views are held on the practical value of the Vi-test. Some authors regard it with disfavour, others think that it may have some value, and a number, more enthusiastic, consider it to be of great importance in detecting typhoid carriers.

The sensitivity and specificity of the tests have been analysed and, taking the prevalence of typhoid carriers into account, it has been possible to calculate the probability of a Vi-reactor being a typhoid carrier.

The figures presented show that the Vi-test should not be used indiscriminately in screening populations for typhoid carriers, but may have limited usefulness in an attempt to trace suspected carriers in a closed group of individuals. A Vi-reactor must be followed up by bacteriological investigation, but the chance of a reactor being a carrier is very small. The demonstration of a carrier among the reactors does not exclude the possibility of another carrier occurring among the non-reactors.

My thanks are due to Dr. A. Wiid, of the South African Council for Scientific and Industrial Research, for statistical advice, and to the Director of this Institute for permission to publish this paper.

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## WORLD MEDICAL ASSOCIATION

### PAKISTAN SEEKS EMINENT MEDICAL SPEAKERS

The Pakistan Medical Association has issued a cordial invitation to the member associations of the world to designate representatives of their organization to present papers at the Sixth All Pakistan Medical Conference to be held in Karachi in November 1960. The exact dates of the Conference will be announced in the near future. The Pakistan Medical Association will provide board and lodging for the medical speakers while they are in Karachi.

The names of delegates to the Conference and the titles of the papers they wish to present should be addressed to: Dr. N. A. Ansari, Secretary, VIth All Pakistan Conference, PMA House, Garden Road, Karachi 3, Pakistan.

EDITORIAL : VAN DIE REDAKSIE

## DR. T. SHADICK HIGGINS

On 30 June Dr. T. Shadick Higgins retired for a second time from spheres of activity in which he has made invaluable contributions. These contributions should be placed on record, for they not only redound to the credit of the medical profession, but also reflect the progress achieved in the public health of our country and the unity of the Medical Association of South Africa.

The citation read at the Adjourned Annual General Meeting of the Medical Association of South Africa held at East London on 28 September 1959, when the Association's Gold Medal for Distinguished Service to the Profession was presented to Dr. T. Shadick Higgins, was published in the *Journal* of 28 November 1959.[1] This gives a summary of his brilliant academic career and also of his achievements as Medical Officer of Health of Cape Town, to which post he was appointed in 1922.

It was our good fortune that the appointment of Dr. Higgins as Medical Officer of Health of Cape Town and Professor of Public Health at the University of Cape Town coincided with the period following immediately on World War I, when overseas public opinion swung in favour of a better and more secure welfare state for everybody. These ideas and opinions also found an echo in our country. Thus the time was ripe and the right man arrived in our midst to help lay a foundation for a practical approach to some of our more urgent problems, such as tuberculosis and promotive health. It was in the field of promotive health especially that Dr. Higgins, with the backing he gained from the Cape Town Municipality, planned and set a pattern of service and organization from which many branches of public health work benefited—maternity services outside hospitals, antenatal and postnatal care, infant welfare, health visiting, and medical advisory services for the promotion of health and the prevention of disease. The beneficial influence of this work was felt and appreciated far beyond the boundaries of the Cape Peninsula.

It was inevitable that this unassuming, erudite, courteous and friendly man would find himself associated at high level with many of the activities within and without the Medical Association of South Africa. Thus, for instance, he found himself playing an important role for varying periods in the Cape Western Branch Council, the Federal Council, the South African Medical and Dental Council, the South African Branch Council and Board of Examiners of the Royal Sanitary Institute, and as the doyen of our Medical Officers of Health (State Medicine) Group, of which he was the President for some fifteen years.

When he retired as Medical Officer of Health of the Cape Town Municipality and as Professor of Public Health at the University of Cape Town he had every reason to look back with satisfaction on his achievements in his adopted homeland, and well may he have rested on his laurels. But this was not to be.

Within a few years Dr. Higgins found himself recalled to duty in the service of the Medical Association of South Africa when, in 1953, he was appointed Editor of the *South African Medical Journal*. It is opportune to place on record that his seven years of service as Editor of the *Journal* coincided with a period of peculiar difficulty in the affairs of the Medical Association and, through his outstanding tact and conspicuous ability, the *Journal* and the Association were able to weather that stormy period so that he could hand over to his successor a sea-worthy ship sailing on an even keel.

It is with a sense of pride that I claim to have had the privilege and honour to have been associated with Dr. T. Shadick Higgins as a colleague and friend ever since he arrived in South Africa, and I am sure I am speaking for the medical profession as a whole when I say to him: We are glad you came, we honour you for what you have done in our country, and we hope you will spend many more happy years in our midst.

Written, at our special request, by
Dr. J. P. de Villiers, of Cape Town.

1. Association Medals (1959): S. Afr. Med. J., 33, 1014.

## SELFMOORD EN POGINGS TOT SELFMOORD

Een van die ernstige probleme waarmee die algemene praktisyn en die personeel van hospitale gereeld te doen kry, is die probleem van die hantering van pasiënte wat pogings tot selfmoord aanwend. En alhoewel hierdie soort pasiënte probleme oplewer wat van algemene mediese belang is, hoort die probleme wat saamhang met selfmoord en pogings tot selfmoord eintlik hoofsaaklik tuis by die voorbehoedende medisyne, die sosiale medisyne, en die psigiatrie.

Ons het reeds al van tyd tot tyd verslae gepubliseer[1,2] wat handel oor verskillende aspekte van dié probleem, soos dit aangetref word in spesifieke gebiede in ons land. Omdat die probleme in hierdie verband egter sulke ernstige implikasies het vir die geneesheer sowel as vir die gemeenskap, sal dit goed wees om sommige van dié implikasies weer van naderby te beskou.

In die eerste plaas is dit belangrik om aan te toon dat daar sekere tipes van pasiënte is wat *veral* in hierdie verband van belang is. Selfmoord of 'n poging tot selfmoord is byvoorbeeld altyd 'n potensiële gevaar in die gevalle van bedrukte pasiënte. Dit sluit pasiënte in met enige vorm van bedruktheid, byvoorbeeld manies-depressiewe toestande, ander endogene bedruktheidstoestande, organiese, reaktiewe, en psigoneurotiese bedruktheidstoestande, en involusiemelancholie.

'n Besonder belangrike groep toestande wat baie algemeen voorkom, maar wat dikwels misgekyk word, is die vae, onomskrewe bedruktheidstoestande van middeljarige persone. Hierdie toestande is besonder verraderlik omdat hulle dikwels ernstiger onderliggende toestande versluier.

Dit is uiters belangrik om nooit 'n egte of 'n pseudo-bedruktheidstoestand gering te skat nie.

Pasiënte wat ly aan ander toestande van ernstige geestesversteuring is ook potensieel selfmoordgevalle. So kom selfmoord of pogings tot selfmoord soms voor by skisofreniese en veral paranoïde pasiënte. Neurotiese pasiënte pleeg selde selfmoord, tensy onderliggende toestande van angs en spanning oorweldigend word. Aan die ander kant kom ‚onsuksesvolle' pogings tot selfmoord, betreklik gesproke, dikwels voor by histeriese neurotiese pasiënte—in welke gevalle die hoofdoel gewoonlik nooit werklike selfmoord is nie, maar 'n poging om aandag op hulself toe te spits.

Pogings tot selfmoord kom natuurlik ook voor by gevalle van vergiftigingsdelirium en ander toestande van ylhoofdigheid waarby die insig en oordeel van die pasiënt tydelik versteur is.

Die vraag of persone wat selfmoord pleeg altyd ‚abnormaal' is en of selfmoord nie ook by ‚normale' persone voorkom nie, is al dikwels bespreek.[3] Alhoewel dit voorkom of hierdie probleem, soos dit nou gestel is, hoofsaaklik van akademiese belang is, het dit nogtans praktiese implikasies. Dit is nie so seer die vraag of persone wat selfmoord pleeg normaal of abnormaal is wat belangrik is nie, as die feit dat daar by sulke persone altyd 'n ernstige mate van persoonlike en maatskaplike disintegrasie teenwoordig is.

Dit is 'n interessante feit dat selfmoord en pogings tot selfmoord somtyds die afmetings van 'n ‚sosiale mode' aanneem. Hierdie soort gedrag is naamlik aansteeklik en dit is bekend dat daar in sekere distrikte soms reekse selfmoordpogings voorkom. Ook wat die metode van selfmoord betref, is daar 'n faktor van ‚aansteeklikheid'.

Dit wil voorkom asof daar in die gemeenskap soos dit op die huidige oomblik saamgestel is, 'n daadwerklike behoefte bestaan aan meer geleenthede vir mense om raadgewing te ontvang op verskillende gebiede van die lewe. Daar is byvoorbeeld baie gevalle waar die geneesheer nie nodig is nie, omdat die betrokke persoon nie siek is nie, en waar die psigiater ook nie nodig is nie, omdat die persoon nie geestesversteurd is nie; maar waar 'n simpatieke raadgewer tog noodsaaklik is. Sulke adviserende dienste word orals oor die land onderneem deur Verenigings soos die Vereniging vir Geestesgesondheid, huweliksburos, kinderleidingsklinieke, die Kerke, ens. Hierdie soort voorkomende dienste behoort uitgebrei te word sodat toestande van ontreddering vroeg behandel kan word voordat hulle ontwikkel tot ernstige vorms van liggaamlike of geestesiekte, of voordat hulle uitloop op die ontwikkeling van wanaanpassing, dranksugtigheid, of pogings tot selfmoord.

1. Walton, H. (1950): S. Afr. T. Geneesk., 24, 933.
2. Klintworth, G. K. (1960): *Ibid.*, 34, 358.
3 Oliven, J. F. (1951): New Engl. J. Med., 245, 488.

# ENVIRONMENTAL BONE DISEASE OF HITHERTO UNDESCRIBED TYPE

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This is a preliminary report of an unusual bone disorder which occurs in a small, localized community and is closely related to fluorosis. 400 Coloured children attend school in Kenhardt, a small town in the north-eastern Karoo in the Cape Province. Of these, 150 come from the 'Rooiblok location' situated ½ mile outside the village. Of the children from this location 30 have been observed to be bow-legged, yet none of those living elsewhere are affected at all.

The condition appears to start when the children are 3 - 5 years old. They complain initially of pain in the legs. The bone deformities become apparent between the ages of 5 and 6 years. These, in the main, consist of bowing of the femora and tibiae (Figs. 1 and 2). The oldest known affected person is 25 years of age, the majority being under 15 years. It is believed by those on the spot that the incidence of this condition is increasing. Many other children in this locality complain of limb pains without showing obvious bony deformity.

Although several cases occur in the same family, affected families are not interrelated. In fact the background and heritage of the families of this 'new' Rooiblok community, established only in 1941, is such as to make an hereditary basis most unlikely.

The children are apparently normal in all other respects apart from the skeletal system. The parents show no obvious bony deformity, but many adults complain of back pain.

Rickets had earlier been suspected on clinical grounds. However, neither specific treatment nor prophylactic therapy have had any obvious effect. From the start the age of onset and curious localization of the condition rendered this diagnosis very unlikely.

In this report we wish to discuss the results of our investigations into the geography, nutritional background, and conditions of this region as well as 2 sample cases investigated in the Department of Medicine, Groote Schuur Hospital.

## THE GEOGRAPHY, ENVIRONMENT AND CONDITIONS OF THE REGION

Kenhardt lies in a hollow between hills. Fig. 3 is a diagram of the area under investigation, *not* drawn to scale. Aerial maps illustrate clearly the tendency for drainage to occur radially towards Kenhardt as a central point. There are, in Kenhardt, two Coloured communities—'Die Lokasie' and 'Rooiblok'. The European settlement lies between the two and is separated from Rooiblok by a river bed which is

*Figs.* 1 and 2. Examples of the clinical deformities found among Rooiblok children attending school in Kenhardt, i.e. bowing of femora and tibiae.

normally dry, but acts as an overflow for the dam (Fig. 3). The European community and 'Die Lokasie' obtain their water supply from several boreholes sunk through rock about 5 miles from the town and lying on the far side of the river bed. The Rooiblok location obtains its water from ghorras—shallow wells sunk to a depth of 10 - 20 feet above the rock.

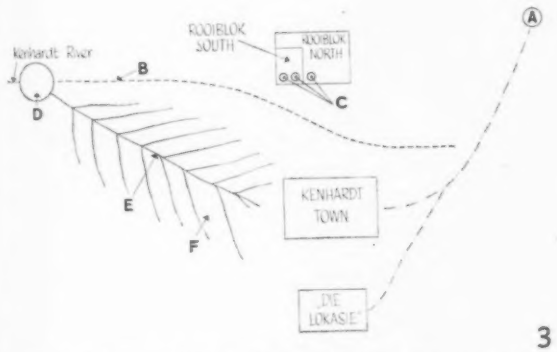



*Fig.* 3. Diagram of Kenhardt area showing the approximate relative situations of the 'dorp' and the locations:
A = Boreholes approximately 5 miles from Kenhardt which supply drinking water to the town and 'die lokasie'.
B = Dry river bed acting as an overflow from the dam.
C = 'Ghorras' from which Rooiblok's drinking water is obtained.
D = Dam built in 1930.
E = Irrigation canal.
F = Area irrigated by dam water.

The river was dammed in 1900 but the wall was washed away soon afterwards. It was rebuilt of earth in 1930. The dam water is used solely for irrigation purposes for the area between the village and the dam.

The Rooiblok settlement has existed for at least 41 years and its water has always been obtained from ghorras. Until 1941, this settlement was a European one, but following a flood it was subsequently resettled by the present Coloured community.

The affected people come from one section of the whole Rooiblok area, that which is closest to the dry river bed which acts as an overflow (Rooiblok South, Fig. 3). One or 2 other cases have come from the area irrigated by the dam, not actually in Rooiblok itself. These people obtain their water supply from ghorras situated near the irrigation canal. The bony disorder occurs only in those children born in Rooiblok South, apart from 2 children (the de Bruin's) who moved into the Rooiblok area at an early age and developed the same condition after some 2 - 3 years.

The diets of the two Coloured communities are believed to be much the same—including meat, milk, and eggs, but with little in the way of green vegetables or fruit. It is certainly highly unlikely that the diets of Rooiblok North and Rooiblok South could differ appreciably from each other. Insecticides are not used on any produce grown. (Certain insecticides are said to cause a rise in the fluorine content of fruit.[1])

## CASES INVESTIGATED AT GROOTE SCHUUR HOSPITAL

*Case* 1

N.M., Coloured female, aged 13 years (Figs. 4 and 5). Bony deformity of legs noted since childhood. No other symptoms. Parents said to be normal. Two siblings have the same condition.

*Figs.* 4 and 5. Case 1 showing clinical deformities.

*General examination:* Afebrile. Thin, rather short (height 4 ft. 3¾ inches). Hypertelorism. The tibiae are bowed anteriorly and the femora laterally. The ligaments are not lax; there are no joint deformities. There is no evidence of old rickets. Teeth show the brown staining of fluorosis with extreme degrees of pitting. There is gross caries at the gingival margins (Fig. 6).

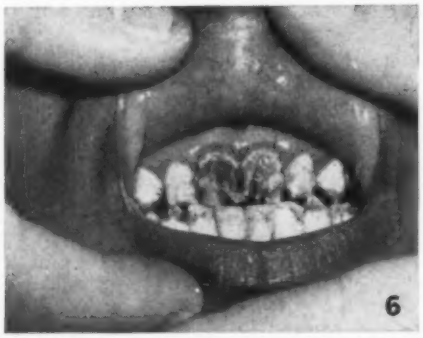


*Fig.* 6. Teeth changes in case 1 showing changes as described in text.

*Cardiovascular system:* Pulse 72/min., blood pressure 115/60 mm. Hg, N.A.D.
*Respiratory system:* N.A.D.
*Gastro-intestinal system:* Liver palpable, 1 finger-breadth below costal margin.
*Central nervous system:* N.A.D.
*Blood picture:* N.A.D.
*Urine:* N.A.D.
*Skeletal survey.* (See Figs. 7 - 9.) Bone age is compatible with chronological age. There is evidence of generalized coarsening of trabeculae with dimineralization and thinning of cortices. In addition there are the following bone changes:
Skull. Thickening of outer table.
Teeth. Defective lamina dura of upper incisor teeth. Areas of erosion in some of the crowns. Central incisors taper in a peculiar fashion towards the apices.
Vertebrae. Bodies somewhat flattened and porotic, showing double transverse lines in each body.
Femora. Bowing laterally with thinning of the cortex and buttressing on the convex aspect.

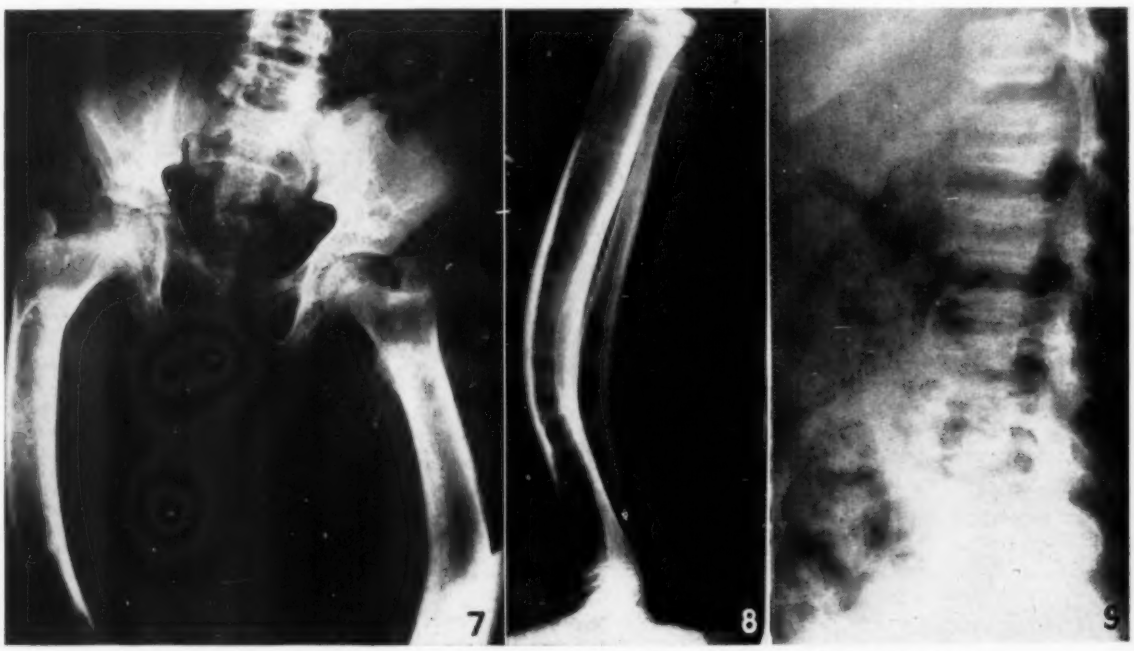
*Other special investigations:* Serum proteins: albumin 4·3 g.% and globulin 3·8 g.%, serum calcium 9·6 mg.%, serum inorganic phosphorus 5·1 g.%, serum alkaline phosphatase 8·2 units (Bodansky-Shinowara), blood urea 18 mg.%, urea and creatinine clearance within normal limits, and Sulkowitch test negative.
Permission for bone biopsy was refused.

*Case* 2

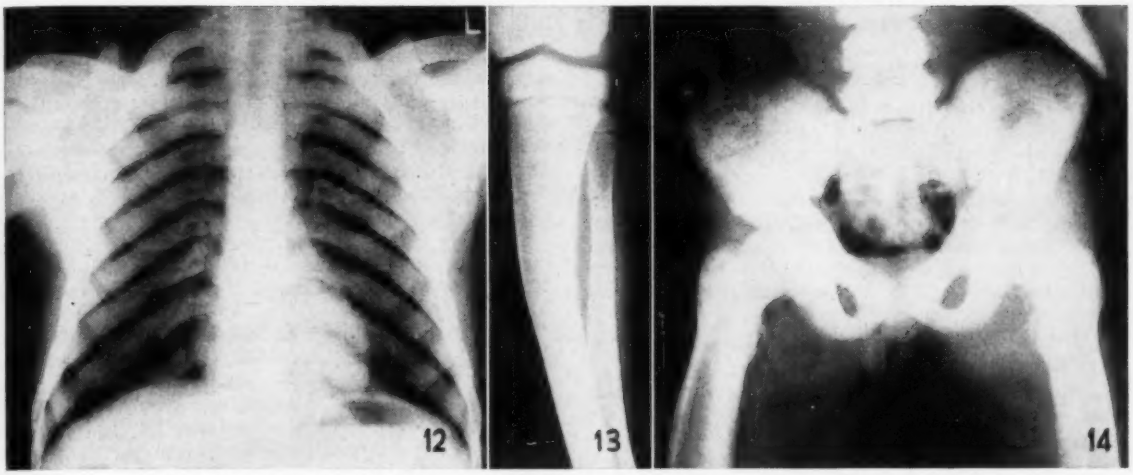
S.S., Coloured male, aged 15 years. (Figs. 10 and 11). Complains of pain in legs on walking or standing. Two older siblings similarly affected.

*Fig.* 10. Case 2 showing clinical deformities.
*Fig.* 11. Teeth changes in case 2.

*General examination:* Height 4 ft. 7½ inches. Pre-pubertal. Teeth showed similar gross caries at the gingival margins with the brown pigmentation and pitting of advanced fluorosis. Anterior bowing of the tibiae (the left more markedly affected than the right). Lateral bowing of the femora. No joint abnormality detected. Other systems clinically normal.
*Blood picture:* N.A.D.
*Urine:* N.A.D.
*Skeletal survey.* (See figs. 12 - 14.) Bone age is compatible with chronological age. Some of the bones showed rather in-



*Figs.* 7 - 9 X-rays of case 1 showing coarsening of trabeculae, demineralization, and deformities described in the text.



*Figs.* 12 - 14. X-rays of case 2 showing the characteristic findings of fluorosis in addition to the bowing of the femora and tibiae.

definite outlines. There was cortical thickening of the long bones and apparent generalized sclerosis as seen in moderately advanced fluorosis, with coarse trabecular pattern and early calcification at the attachments of the ligaments and muscles. The femora and tibiae were bowed in a similar manner to case 1.

Serum calcium, inorganic phosphorus, blood urea, serum potassium, sodium, chloride, and bicarbonate were all within normal limits.

*W.R. and Berger:* Negative.

*Urea clearance:* Normal.

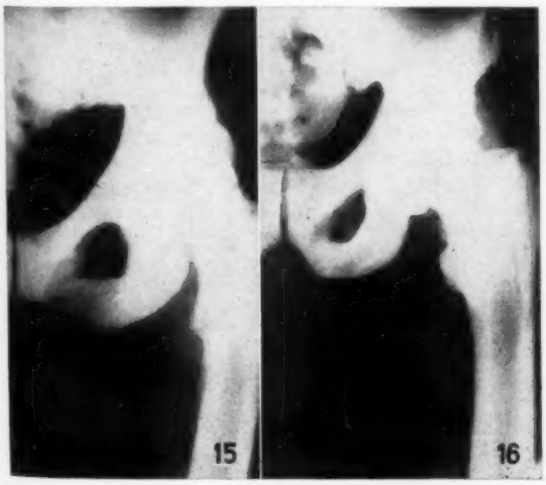
24-hour urinary calcium was 41 and 63 mg. per 24 hours. 24-hour urinary phosphorus was 470 and 1,170 mg. per 24 hours. 24-hour urine for fluorine analysis revealed 6·6 mg. in a volume of 2,350 c.c.

*Bone biopsy* (greater trochanter): 'Specimen consists of a single piece of cartilage-covered bone 4·5 cm. in diameter.

Histology shows cessation of bone formation at the junction of bone and cartilage. Bone trabeculae vary in size and thickness, but most are somewhat cellular and a few contain small islands of cartilage in the midst of the bone. Occasional groups of active osteoclasts and osteoblasts are seen and cement lines are prominent but have not resulted in a Paget's-like mosaic. Some basophilic stippling of bone matrix appears to be an artefact.

Neither excessive bone resorption nor woven bone formation, nor unduly wide osteoid seams were observed' (Dr. J. A. H. Campbell).

*Fluorine analysis of bone:* 8,500 p.p.m. fluorine.



*Figs.* 15 and 16. X-rays of parents of case 2 showing the classic characteristics of fluorosis.

X-*rays of Parents of Case* 2

The pelvis and upper ends of the femora of both adults (Figs. 15 and 16) present virtually the same appearance. There is gross generalized sclerosis, with obliteration of the trabecular pattern and thickening of cortices of the femora. Irregular outlines of the bones are produced by calcification in several ligamentous and muscular attachments.

## FURTHER INVESTIGATIONS OF SAMPLES FROM THE AFFECTED AREA

### *Water*

On investigating *water samples* for their *fluorine content*, the following findings were recorded: Rooiblok South 7·4 p.p.m., Rooiblok North 6·0 p.p.m., and 'dorp' (village) water 2·6 p.p.m. 'Dorp' water is also supplied to the other Coloured location. The critical level of fluorine content, with respect to bone disease, probably lies in the region of 2 p.p.m.

### *Animal Bone*

The fluorine content of animal bone (sheep from Rooiblok location) was found to be 145 p.p.m. The results were calculated on the bone ash. Monier-Williams[6] gives the normal content of leg of lamb as 28 - 36 p.p.m. A live chicken was obtained from the affected area. Chickens are said to be abnormally resistant to fluorosis. When affected they develop a stiff-legged gait with ankylosis of the knee joints. The specimen bird was, unfortunately, a very young one. It was submitted to Dr. W. A. J. du Plessis of the Blue Cross Veterinary Hospital, Newlands, Cape, who kindly observed the bird. No abnormality could be found. It is still under observation.

### *Investigations in Progress*

A further radiological and photographic survey of more affected persons and also 'controls' in the neighbourhood

is being organized, and further analyses of water and soil from the Kenhardt area are under way.

## DISCUSSION

We have described a bone disorder occurring in a sharply delimited community and locality. There is good evidence that hereditary factors do not play any part.

It seems, therefore, that the causative factors must be environmental. Superficially the X-rays of the 2 young patients seen in this hospital suggest 2 different conditions, because the skeleton of one of the patients is more dense, and that of the other less dense than normal. More careful examination, however, indicates a basically similar pattern, for which the evidence is:

1. The clinical similarity of these 2 cases and others from the same locality.

2. Although the radiological consistency of the bone differs, the coarsening of the bony trabeculae and the type of gross deformity is similar in the 2 cases.

3. The abnormalities of the teeth were also the same in both patients.

It seems that this may be the same disorder seen at different stages of its development.

It is known that fluorosis is endemic in the Kenhardt area.[1] The teeth of people in this area show the mottling and pitting of fluorosis. In addition the caries which occurs at the gingival margin in gross fluorosis is common throughout the district. While a minimum fluorine content of drinking water is necessary and protective to teeth, the severe pitting found in advanced fluorosis may predispose to caries.

The X-ray findings in case 2 are quite characteristic of fluorosis, except for the altered shape of some of the bones which indicates an unusual degree of softening at some stage.

Classically, fluorosis manifests radiologically as a marked sclerosis, at first with accentuation of the trabecular structure. Later apparent coalescence of trabeculae gives a picture of generalized uniform increased density of the bones, which appear hazy in outline, chalky, and structureless. The regions of the spine and pelvis are most affected. In advanced cases, calcification of ligaments can occur.

The end-stage is that seen in the pelves of the two adults as described above. Case 2 illustrates the earlier phase of fluorosis, although the bowing of the bones is not typical of this condition. Case 2 could be said to show a radiological picture somewhere between the picture of case 1 and that of his own parents.

There is little reference in the literature to softening or bowing of the bones in fluorosis. In his book on fluorine intoxication Roholm[2] discusses 3 clinical types: (1) Teeth changes, (2) the osteosclerotic form, and the (3) 'osteomalacic' form (also seen in animals) in which the bones are 'reduced in strength' and bone atrophy and defective calcification occur.

Weinmann and Sicher[4] in their discussion on fluorosis point out that the findings in spontaneous and experimental fluorosis are, in part, contradictory. The bones of human patients are usually diagnosed as sclerotic, while osteoporosis and 'osteomalacia' have been reported in diseased animals and in experiments.

These authors suggest that the radiological examination and the weights of bones may lead to a false impression, since X-rays may not differentiate between true osteosclerosis and the apparent increase of calcium content of bone due to calcification of the soft tissues, e.g. endosteum, periosteum, blood vessels, and ligaments. Their study of the histology had led them to postulate that the presence of fluorides causes rapid resorption of bone commencing in the marrow cavity, without inhibition of new-bone formation at the periosteal surface.

In young individuals the resorption is very rapid and the new bone is immature in type, consisting of loosely arranged spongy bone as opposed to the mature lamellated bone in older persons.

In a survey of fluorosis in Nalgonda, India, only dental changes in the children were found.[3] Shortt *et al.*[5] state that the bone changes take 30 - 40 years to develop. Poor nutrition, high altitude, and excessive heat were thought to be factors accounting for the earlier onset in the Nalgonda series. (Their youngest case with bone changes was 21 years old.)

Skeletal changes are rarely found in the USA in spite of fluorine levels of 8 p.p.m. in the water of certain regions.[4]

There can be no doubt that our young patients demonstrated fluorosis, in common with their elders. Despite the work quoted above, no mention of softening or bending of bones in clinical human fluorosis has been found, and in any event the clinical manifestations are rare in childhood. Furthermore, the concentration of fluorine in the drinking water is no higher than in many other places in the world, from where no similar phenomena have been reported.

Taking all the unusual aspects of our cases into account, perhaps we can postulate:

1. That fluorine is only one of the factors, though probably the most important, and

2. That in combination with either some other deficiency or a secondary factor, as yet unknown, fluorosis is occurring in an enhanced, accelerated, and unusual form.

We are presenting this problem in the hope that those of our colleagues, who may have come across similar cases in other areas, will contribute their experience and ideas to the solution.

## SUMMARY

An unusual bone disorder is reported which occurs in a small localized community in the outskirts of Kenhardt. It is related to fluorosis.

We should like to thank Dr. J. G. Burger, Superintendent, Groote Schuur Hospital, Observatory, Cape, for his interest in this study; Prof. J. F. Brock, who arranged for us to undertake this investigation; Dr. L. Werbeloff; Dr. W. Rosenblatt, consultant dental surgeon to Groote Schuur Hospital; Dr. J. A. H. Campbell for the histological opinion; Dr. L. Been of the Union Health Department, for putting so many facilities at our disposal, Mr. N. Penny of the Government Laboratories for the chemical analyses, Mr. B. T. A. Todt for the clinical photographs and Mr. E. R. Levick for the diagram.

This work was supported in part by the South African Council for Scientific and Industrial Research, in a grant to the Endocrine Research Group of the University of Cape Town.

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6. Monier-Williams, G. W. *Trace Elements in Food.* New York: John Weley.

## ADDENDUM

The following is an analysis of a specimen of water from a well in Rooiblok South. This analysis was done in the Department of Agricultural Technical Services Scientific Laboratory in Johannesburg.

'Report on the analysis of a sample of water taken from No. 2—Rooiblok-Kenhardt—Open Well. Received on 22 March 1960. Colour: White. Sediment: moderate. Muddiness: nil. Conductivity: 2,200

IN PARTS PER MILLION PARTS OF WATER

| | |
|---|---|
| Total solids 1,480 | Saline ammonia 0·04 |
| Solids in suspension — | Albuminoid ammonia 0·24 |
| Chlorine 500 | Oxygen absorbed (4 hours at 27°C) 0·05 |
| Sulphur - oxide 137 | Total hardness 430 |
| Nitrogen as nitrate 10 | Permanent hardness — |
| Nitrogen as nitrite *nil* | Alkalinity (as calcium carbonate) 420' |
| Fluorine 4·8 | |

# GENERALIZED CRYPTOCOCCOSIS WITH OSSEOUS INVOLVEMENT

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Cryptococcosis or torulosis has been prominent in the medical literature in recent years, and its protean manifestations have been well reviewed. The pulmonary, osseous and central nervous systems are most frequently involved. In the acute fulminating cases a meningo-encephalitis is nearly always present, and the outcome is almost invariably fatal. Miliary involvement of the lungs may occur in the disseminated disease. In the more chronic cases pulmonary involvement is frequent. Pulmonary lesions may be indistinguishable from tuberculosis, or may be present as solid masses resembling tumours. Tumours of the lung due to *Cryptococcus histolyticus* (*neoformans*) were reported by Dormer and his collaborators in 1945[1] and 1947.[2] Osseous lesions tend to occur when the infection is generalized. Prior to a report of 3 cases by Collins[3] in 1950, there had been 17 instances of bone involvement in a review of over 200 cases reported in the literature. Multiple bone lesions were present in a case report by Wiener[4] in 1951. In an analysis of 21 cases by Wolfe and Jacobson[5] in 1958, there were 3 cases of osseous involvement. In 1958 Hilbish[6] stated that bone involvement occurs in approximately 10% of patients with the disseminated disease.

The lesions are characteristically osteolytic. Progression and regression are slow. Comparison with bone lesions in other mycoses,[3] notably blastomycosis and coccidioidomycosis, stresses that periosteal proliferation is a frequent finding in the latter. All the mycotic lesions are destructive, with little reactive sclerosis. Predilection for bony prominences is a feature of mycotic lesions, as opposed to other infections with multiple bone involvement. Abscess formation is frequent at the site of bone lesions. The pus is characteristically mucoid or gelatinous.

Association of cryptococcosis with sarcoidosis and Hodgkin's disease is mentioned by Collins.[3] Coexistent tuberculosis is uncommon.

Up to the time of the discovery of amphotericin B, no satisfactory treatment was known, and cases of generalized infection were very frequently fatal.

### CASE REPORT

P.S., Bantu male aged 14 years, was admitted to King George V Hospital on 5 January 1959. He was transferred from McCord's Zulu Hospital, Durban, where he had been treated for typhoid fever since 25 September 1958. The diagnosis of typhoid fever was confirmed by positive stool culture. He was persistently febrile on subsidence of the typhoid fever. X-ray of the chest on 29 October 1958 (Fig. 1) showed miliary nodulation, which was regarded as tuberculous and treated with izoniazid, 200 mg. *t.d.s.* and streptomycin, 1 g., twice weekly. Chest X-ray on admission to King George V Hospital showed clearing of the nodulation. Antituberculous treatment was continued.

On 9 January 1959 he developed a swelling of the left upper eyelid, which appeared to be a sebaceous or meibomian cyst. On 20 January he complained of pain in the left ankle and gave an erroneous history of trauma. The ankle became swollen. X-ray on 11 February showed an erosion in the os calcis, which was regarded as a tuberculous osteitis. A plaster cast with walking sandal was fitted. The swelling in the left orbit gradually increased in size, became fluctuant, and discharged muco-pus spontaneously on 20 March. At this time he was found to have a cervical adenitis.

On 11 April a foul-smelling discharge from the left ankle was observed. On removal of the plaster there were 2 large erosions of the skin with a sloughing base and a watery discharge. Repeat X-ray on 15 April (Fig. 3) showed increase in extent of erosion of the os calcis.

At this time a painful swelling of the left side of the chest developed, and X-rays on 15 April showed an osteitis of the 9th rib at the site of the swelling. Shortly after this a swelling over the xiphisternum appeared. Swabs taken from this yielded no organisms on culture. Swabs taken from the heel showed haemolytic *Staphylococcus albus*, sensitive to streptomycin, achromycin, chloromycetin, ilotycin, furadantin and aureomycin.

X-ray of the cervical spine on 23 April revealed lesions of the 4th and 5th vertebrae, consistent with tuberculosis. A Mantoux test at this stage was positive.

The patient had been on continuous antituberculous treatment since admission. The progression of the erosion of the os calcis and the development of other foci of bone infection threw grave doubt on the tuberculous aetiology of the disease. As he had had typhoid before transfer to King George V Hospital, the possibility that the bone infections might be caused by *Eberthella typhosa* was considered. Blood culture, however, was sterile. Chloromycetin therapy was commenced empirically, but resulted in no improvement.

On 11 May pain over the left ilium developed. X-ray showed an erosion in the anterior superior iliac spine.

In smears from the discharging heel, mycelial elements were seen on staining. The patient was put on potassium iodide in increasing doses up to 90 gr. daily and mycostatin, 2 tabs 4 times a day.

Repeat X-ray on 27 May showed further erosion of the os calcis, with decalcification of the foot bones. The eroded area in the left ilium appeared slightly larger. The clear-cut focus of infection in the 9th rib had not changed, but periosteal reaction was evident (Fig. 2). In the cervical spine (Fig. 4) there was progression of the infective process, but less prevertebral swelling.

The first laboratory reports (culture) indicated the growth of a fungus of candida type, but as the colonies grew it became evident that they resembled *Cryptococcus neoformans*. A rich growth of

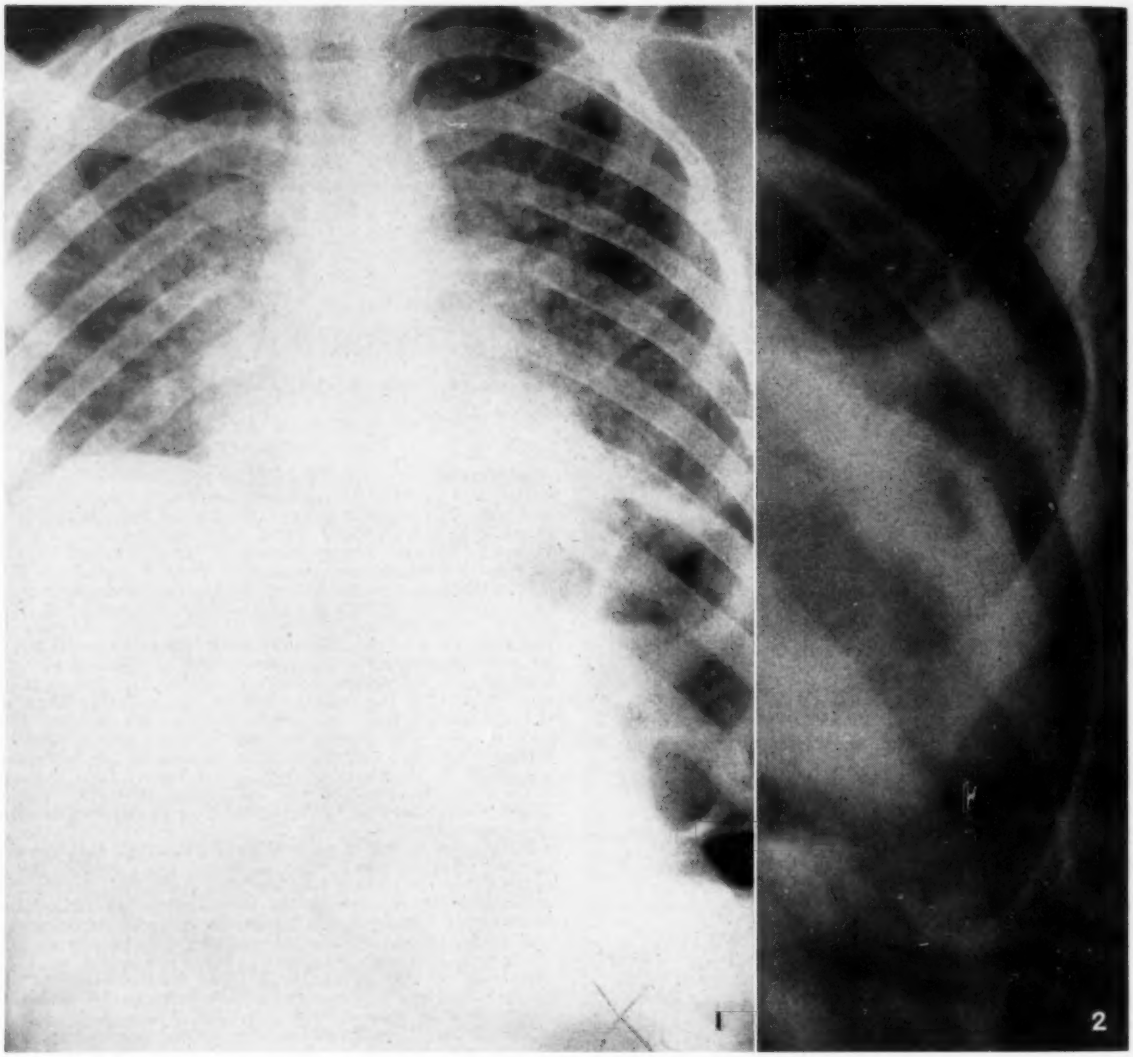

*Fig.* 1. Chest, 29 October 1958, showing miliary nodulations in lungs. *Fig.* 2. Ribs, 27 May 1959, showing clear-cut erosion in 9th rib, with periosteal reaction.

this organism in pure culture was obtained. Mice were inoculated and positive growth was obtained. In view of this finding, therapy with amphotericin B was commenced on 3 June. A polythene catheter was inserted into the inferior vena cava, *via* the right saphenous vein, and a continuous drip containing amphotericin B was started with a concentration of 0·25 mg. per kg. for the first day, increased to 0·5 mg. the next day, and then up to 0·75 mg. per kg. It was kept at this level. There were no reactions for the first 6 days. Then a steady pyrexia commenced and became persistent, and the dosage had to be reduced to 0·25 mg. per kg. At this level there was practically no reaction, and the dosage was maintained until 19 June.

A blood count on 13 June was as follows: Hb. 11 g.%; wbc. 15,000 per c.mm.; rbc. showed hypochromia and anisocytosis; platelets appeared slightly increased; polymorphs showed a marked shift to the left; two metamyelocytes were seen; differential count—neutrophils 93%, lymphocytes 6%, mononuclears 0, eosinophils 1%.

The blood culture for fungi was reported as negative on 27 June.

A great improvement followed clinically on this treatment. On 18 June repeat X-ray of cervical spine, ribs and ankle showed a healing process. Follow-up X-rays on 18 August of cervical spine and on 16 September of ankle and ribs showed progression of healing. X-ray of chest showed practically clear lung fields.

The patient was discharged on 26 October 1959.

## DISCUSSION

There were several factors which contributed to the difficulty in making a diagnosis in this case.

The diagnosis of typhoid fever before admission to this hospital seems to be established. The miliary lung infection was indistinguishable from miliary tuberculosis, and had to a large extent resolved on admission, apparently in response to anti-tuberculous treatment. In retrospect it appears probable

*Fig.* 3. Left ankle, 15 April 1959, showing erosion in os calcis.

*Fig.* 4. Cervical spine, 27 May 9159, showing erosion of anterior surfaces of C4, 5 and 6 on adjacent surfaces of C4 and 5.

that the miliary lung involvement was not tuberculous, but an early manifestation of systemic cryptococcus infection. The subcutaneous lesion of the left orbit was apparently another manifestation of generalized infection.

The bone lesions which followed the lung and subcutaneous infections in no way differed from those of tuberculous aetiology. It was the successive development of these, and their progression while on antituberculous treatment which made it appear unlikely that tuberculosis was the infective agent. The Mantoux test, however, was positive.

The subsequent behaviour of the ankle infection was not like tuberculosis. It was from the swabs of this lesion that the diagnosis of *Cryptococcus neoformans* infection was established.

The patient's condition deteriorated until specific antifungus treatment was initiated and, from then onwards, a steady improvement was manifest, and maintained until he was fit for discharge.

SUMMARY

The manifestations of systemic cryptococcus infection are briefly reviewed, particularly in relation to pulmonary and osseous involvement.

A case is reported in which the initial features of generalized infection were pulmonary and subcutaneous.

Multiple osseous lesions followed. These were all osteolytic in nature, and resembled tuberculous infection radiologically.

The development of successive lesions while the patient was on anti-tuberculous treatment led to the investigations which eventually established the diagnosis.

A satisfactory response to treatment by amphotericin B is described.

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# 'N MOONTLIKE NUTTIGE MUSKIET, *THEOBALDIA LONGIAREOLATA* MCQ*

J. J. STEYN, B.Sc., PH.D., F.R.E.S., *Navorsingsinstituut, Tzaneen, Transvaal*

Dis algemeen bekend dat die huisvlieg, *Musca domestica* L., 'n reeks van malaria-muskiete, ander muskietsoorte, sowel as 'n verskeidenheid van verdere insekte van mediese belang, gedurende die afgelope 15 jaar weerstand ontwikkel het teen die moderne insekdodende middels. Derhalwe hou navorsers hulle oë oop vir moontlike suksesvoller metodes om insekte-vektors van mens- en diersiektes te beheer, bv. deur gebruik van die natuurlike vyande van die vektors. Ons gegewens wat aandui dat *Theobaldia longiareolata* 'n nuttige muskiet is in die stryd teen die kosmopolitiese huismuskiet, *Culex fatigans* Wied., word dus aangekondig.

*Autogenie*

Gewoonlik moet 'n muskietwyfie bloed van die een of ander gasheer verkry om haar eiers te ontwikkel. Muskiet-mannetjies leef egter van plantnektar, en hulle suig nooit bloed nie.

Maar nou is daar 'n paar uitsonderinge, bv. die wyfies van *Toxorhynchites brevipalpis* Robineau-Desvoidy[1] en *Aëdes taeniorhynchus* Wied.[2] het nie bloed nodig vir eierontwikkeling nie. Hierdie verskynsel is bekend as autogenie, en kom volgens my ondervinding deurgans voor by *Theobaldia longiareolata* in Suid-Afrika.

Gedurende muskietopnames sedert 1950 in Natal, die Transvaal, die Vrystaat en die Kaapprovinsie, en ook gedurende ons slenkdalkoors-ondersoeke, sowel as by die identifikasie van *T. longiareolata* monsters wat deur malaria-veldpersoneel, gesondheidsinspekteurs en ander versamelaars verskaf is, het ek nooit 'n enkele *T. longiareolata* wyfie met 'n bloedmaal in haar pensie teengekom nie.

Ook wanneer honderde van hierdie muskietsoort herhaaldelik in hoenderhokke gevind is, was daar geen wyfie wat 'n bloedmaal geneem het nie.

Op Vaalhartz is perde, beeste, en skape, en ook Blanke en nie-Blanke persone nooit gebyt deur longiareolata by drinkbakke wat byna swart was van larwes, papies en volwassenes van hierdie soort nie, alhoewel ons waarnemings dikwels dwarsdeur die dag en nag gedoen is vanaf Julie 1951 tot Februarie 1952.

Selfs wanneer honderde volwasses uit larwes en papies op 'n ingegaasde stoep, waar ek gewerk en geslaap het, op Vaalhartz geteel is, is ander Blankes en ekself nooit gebyt nie. Nooit het dit eers gebeur dat Theobaldia op ons kom sit om bloed te suig nie; en nooit is enige bloed in die wyfies opgemerk nie.

Gedurende Desember 1956 was skoolkinders op Wepener baie verbaas dat ek nie wis dat Wepener ‚muskieteloos' is nie. En toe ek langs hulle speelplek 'n swart massa van honderde Theobaldia larwes en papies in 'n drom by die waterkraan vind, wou hulle nie glo dat dit werklik onvolwasse muskiete was nie.†

Muspratt[4] skryf: ‚The adult females seldom attack man.' Aan ons gegewens getoets, is hierdie bevinding nuttig, want by wyse van uitsondering bewys dit die verskynsel van autogenie nog verder.

Naas die afwesigheid van bloed in Theobaldia se pensies, en autogenie, is ons belangstelling in ongevoede, gevoede, halfdragtige en voldragtige *Anopheles gambiae* en ander Anophelines reeds beskryf.[5,6,7]

Die verskynsel van autogenie interesseer ons ook verder omdat ons gedurende slenkdalkoors-ondersoeke altyd gevind het dat *T. longiareolata*, *Anopheles squamosus* Theo en *Aëdes caballus* Theo aanwesig was: en autogenie Theobaldia as 'n moontlike slenkdalkoorsvektor uitgesluit het.

*Broeiplekke en Kompetisie met die Huismuskiet*

Muspratt[4] skryf oor longiareolata: ‚It breeds in veld and river-bed pools including rock pools, and the writer found it to be the predominant species at Springbok in Namaqualand at the beginning of the short rainy season. In the Orange River Valley of the northern Cape Province it seems to breed chiefly in pools in the river valley during the winter months, and from there probably spreads to the surrounding Karoo country during the limited rainfall. Larvae are also found in water tanks and in cattle and sheep troughs, which may help to maintain the species in some arid districts . . . The eggs of this genus is apparently not resistant to dessication'.

Hopkins[2] noem as verdere broeiplekke: vaatjies, diptenks, 'n bokseil met water; ou oliedromme, en soms vore en poele. Ook die Marra Gebergtes bo 6,000 vt. in die Soedan, en klein poele langs strome is gewilde broeiplekke. Rotspoele, sowel as groter, rotsige sypoele met staande water dien ook dikwels as broeiplekke, asook vars water en brak water van die soutmeer te Dereiba. Broeiplekke kom selfs voor in balies op die Bizen Berg, omtrent 7,800 vt. bo seespieël.

Behalwe in die bogenoemde verskeidenheid van broeiplekke, het ons longiareolata in digte massas van larwes en papies gevind in koue grasbegroeide vleipoele gedurende wintermaande, in groot oop modderpoele langs paaie, in grasbegroeide poele langs paaie, in sinkdamme en sementdamme, in grasbegroeide ou leiwatervore, by fonteine, langs gronddamme en in waterpoele, in populierbosse en in Port Jacksonbosse, in 'n swembad, in voëlbaddens, visdammetjies in blomtuine, sementbakke by kwekerye, buitebande, houtbalies, konkas, blikke sowel as in sink- en sement-reënwatertenks.

In my ondervinding was die deurgaans suiwer populasies of feitlik suiwer populasies 'n opvallende kenmerk orals waar hierdie soort raakgeloop is. Van verdere belang is die feit dat die genoemde broeiplekke van longiareolata juis altyd dié van die gewone huismuskiet, *Culex fatigans* is. Ons bevindings was so konsekwent dat ons moet besluit dat longiareolata 'n doeltreffende en suksesvolle konkurent van die huismuskiet is.

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* Gepubliseer met toestemming van Die Sekretaris van Gesondheid, Pretoria.

† Onwillekeurig het dit my laat dink aan die entomologiese reus, Jacobus Faure, aan wie die Karooboere vertel het dat die baie kleiner veldsprinkaan met die groot bruinsprinkaan paar na uitbreke van die bruinsprinkaan; en hoe sy navorsing toe die fase-teorie wat ook vir ander swermsprinkane deur die wêreld bevestig is, gelewer het. En ook aan die ou leerstelling dat blywende entomologiese ontdekkings gemaak kan word deur navorsing aan die hand van die Bybel, sprokies, legendes, spreekwoorde van verskillende nasies, en die waarnemings van boere.

Wanneer daar tog soms enkele fatigans larwes tussen massas longiareolata larwes en papies gevind is, was dit waarskynlik dat die aktiwiteite van hierdie groter soort waarvan die sterker, lywiger larwes se volume seker 6 keer dié van fatigans is, die kleiner soort maklik verdring. Ook word volwasse fatigans, wat uit die papies kom en moet rus sodat die vlerke, bene en liggaam hard word voordat hulle kan wegvlieg van die papiervelletjies af, seker heel dikwels deur die groter longiareolata larwes se bewegings, verdrink.

Vermoedelik was dit longiareolata larwes wat ek as kind met my hand in ons reënwatertenk op Zastron gevang het. En te oordeel na die ondervinding van my 9 broers en susters wat saam met my langs hierdie reënwatertenk, muskietevry opgegroei het, en nadat massas Theobaldia in Desember 1956 op Zastron gevind is, moet ek Wepener se kinders gelyk gee en byvoeg dat Zastron, nes Wepener, ‚muskieteloos' is. Eers op Petrusburg, in die Oranje-Vrystaat het ek muskietbyte leer ken.

Volgens ons waarnemings broei longiareolata blykbaar dwarsdeur die winter in die Kaapprovinsie, die Vrystaat, die Transvaal, en Natal en oorwinter ook as volwasse muskiete. Weens die beskrewe broeigewoontes het hierdie muskietsoort dus reeds 'n groot voorsprong bo die huismuskiet.

Van verdere belang is weereens die uitsonderlike lengte, breedte, volume, en beweegkrag van Theobaldia larwes wat hulle in staat stel om in groot getalle in sinkdamme, sementdamme, en ook in diep poele, te leef waar hulle ten spyte van die diepte en windveroorsaakte golfbeweging tog suksesvol op organiese materiaal op die bodem en wateroppervlakte voed. In sulke broeiplekke het ons nooit die kleiner en swakker huismuskiet se larwes gevind nie.

Behalwe in water met 'n lae gemiddelde organiese inhoud, teel Theobaldia ook flink in houers en poele wat stampvol van verrottende hooi of blare is. En volgens ons waarnemings druk longiareolata ook in hierdie broeiplekke die huismuskiet suksesvol uit.

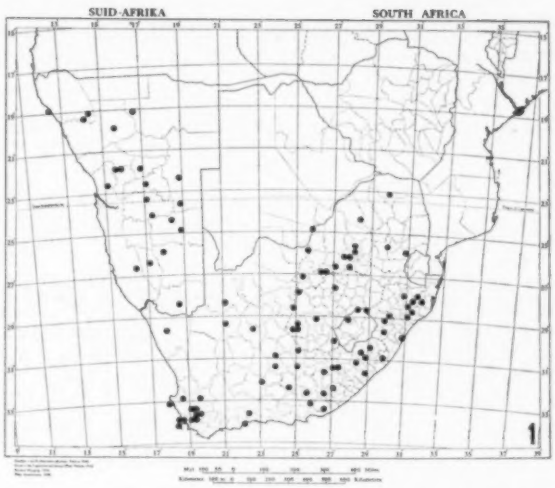
Die feit dat *T. longiareolata* autogeen is, gee hierdie soort nog 'n verdere uitgesproke voorsprong bo die kosmopolitiese huismuskiet wat bloed van 'n gasheer moet neem om eiers te ontwikkel.

Ons meen derhalwe dat dit nuttig mag wees om longiareolata se biologie na te vors en metodes te ontwikkel om sy suksesvolle kompetisie teen die huismuskiet verder te verhoog en sodoende die mens, sowel as sy huis- en plaasdiere, beter te beskerm.

*Voorkoms*

Volgens beskikbare gegewens wat goedgunstiglik deur die Mediese Ekologiesentrum, Johannesburg, verskaf is, word die voorkoms van *T. longiareolata* in Suid-Afrika in Afb. 1 aangetoon. In sy verspreiding deur Suid-Afrika is dit van belang om daarop te wys dat hy in al die 15 klimaatstreke dwarsdeur ons land bekend is; ook is hy een van die paar van ons 156 Suid-Afrikaanse muskietsoorte wat die meeste dwarsoor die land voorkom.

Hierdie muskiet is ook reeds gevind in die Soedan (Dongola Provinsie, Damer, Erkowit, Khartoem, en Medani), Brits Soemaliland, Abessinië, en is wydverspreid in die Middelandse See-gebied van die Azores, die Kanariese Eilande en Madeira, deur Spanje na Macedonië, Persië en Noord-Wes Indië, sowel as deur Morokko en dwarsdeur die noordelike Sahara na Egipte en Palestina.[1] In Oeganda het ek hierdie soort




*Afb.* 1. Die voorkoms van *T. longiareolata* in Suid-Afrika. (Die kaart is herdruk ingevolge die Staatsdrukker se outeursvergunning nr. 2752 van 3 Maart 1960.)

ook in 1945 gevind. Dis derhalwe duidelik dat hy baie wydverspreid is.

MOONTLIKE NAVORSING

Omdat *T. longiareolata* naas die autogene *Toxorhynchites brevipalpis* en *Aëdes scatophagoides* Theo. een van ons grootste muskiete is, het hy 'n suigmond wat baie langer is dan dié van die gewone huismuskiet, *Culex fatigans*. Dus verwag ons dat albei geslagte van longiareolata nektar kan bykom in die blomme van sekere plantsoorte waarvoor die suigmond van die ver kleiner fatigans te kort is. Dit mag dus nuttig wees om deur middel van radio-aktiewe isotope vas te stel watter plantsoorte gebruik kan word om dwarsdeur die jaar nektar te lewer net vir longiareolata, terwyl fatigans dit nie kan bykom nie, om sodoende die autogene longiareolata se voorsprong verder te vergroot vir die biologiese bestryding van huismuskiete ter beskerming van die mens, sy huis- en plaasdiere.

Hierdie pleidooi word verder ondersteun deur die ou bekende beginsel van Ullyett dat dit soms net nodig is om die natuurlike sterfte van 'n insekteplaag op die een of ander swak plek in sy lewensgeskiedenis kunsmatig met 2 of 3 % te verhoog, om die prentjie in die mens se guns te verander.

SUMMARY

Observations have revealed that a large mosquito, *Theobaldia longiareolata* is autogenous, and does not take blood meals. It is considered that this phenomenon explains why the towns of Wepener and Zastron are regarded as 'mosquito-free'. The known breeding places of this mosquito are discussed. Findings disclose that it is probably a very successful competitor of the cosmopolitan house mosquito, *Culex fatigans*. Its known distribution is recorded. A plea is made for further research into the biology of longiareolata to devise methods for augmenting its competition against the house mosquito.

Ons bedank graag mnr. D. H. S. Davis, Hoof, Mediese Ekologiesentrum, Johannesburg, vir die voorkomskaart wat hy goedgunstiglik opgestel het. Ook betuig ons ons werklike dank aan al die belangstellende versamelaars wat die moeite gedoen het om materiaal te versamel.

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## PHARMACEUTICAL NEWS : FARMASEUTIESE NUUS

### WELLCOME TRUST GRANT TO THE MASSACHUSETTS GENERAL HOSPITAL, BOSTON, USA

The Wellcome Trustees have made a grant of £50,000 to the Massachusetts General Hospital, Boston, for the establishment of a Henry S. Wellcome Research Chair in Medical Science. The professor will be chosen by a special committee of the Massachusetts General Hospital, Harvard University and the Massachusetts Institute of Technology, from a number of workers who have already achieved a high scientific reputation for their researches. The appointment will be made for 10 years.

This grant was announced in Boston on 7 June, in relation to the preparations for the 150th anniversary of the founding of the Massachusetts General Hospital which falls next year.

This is the largest single grant to be made by the Wellcome Trustees to a medical research centre in the United States. In 1956 they made a grant of nearly £25,000 to the University of Pennsylvania to provide a Wellcome Associate Research Professorship of Anesthesiology for 5 years, and earlier in the present year they made a grant of £15,000 to construct a laboratory for cardiovascular research within the Department of Medicine at Johns Hopkins Hospital, Baltimore, USA.

## PASSING EVENTS : IN DIE VERBYGAAN

*South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting.* The next meeting will be held on Monday 25 July at 5.10 p.m. in the Institute Lecture Theatre. Major J. Horak will show the film 'Vertical frontier', which was made in cooperation with the United States Air Force.

* * *

*Dr. Roy Morris,* physician, of Johannesburg, has recently returned from a trip overseas where he visited medical units in New York, Boston and London, and has now resumed practice.

* * *

*Southern Transvaal Branch (M.A.S.A.).* The Annual General Meeting of the Southern Transvaal Branch will be held at Medical House, 5 Esselen Street, Hillbrow, Johannesburg, on Tuesday 26 July 1960 at 8.15 p.m. Prof. P. V. Tobias, Professor of Anatomy at the University of the Witwatersrand, will address the meeting on 'Some aspects of human heredity'. Senior students are invited to attend this meeting.

* * *

*Prof. James T. Louw,* Professor of Obstetrics and Gynaecology at the University of Cape Town, is at present on an overseas visit. On 13 July he was one of the main speakers at the Scientific Meeting held to mark the opening of the new building of the Royal College of Obstetricians and Gynaecologists in Regents Park, London.

* * *

*Members are reminded* that they should notify any change of address to the Secretary of the Medical Association of South Africa at P.O. Box 643, Cape Town, as well as to the Registrar of the South African Medical and Dental Council, P.O. Box 205, Pretoria. Failure to advise the Association will result in non-delivery of the *Journal.* This applies to members proceeding overseas as well as those who change their addresses within the Union.

* * *

*Presentation to Dr. T. Shadick Higgins.* As members of the Association will know, Dr. Shadick Higgins left the service of the Association at the end of June.

After many years as a member of Federal Council and a member of the South African Medical and Dental Council, Dr. Higgins, during his retirement from the post of Medical Officer of Health of Cape Town, was appointed Editor of the *South African Medical Journal* in 1953. On the appointment of Dr. A. P. Blignault as Editor in 1959, Dr. Higgins continued to assist in the work of the *Journal* as Associate Editor.

His many friends on the Head Office and Journal Committee and in the Cape Western Branch made a presentation to him at a Dinner which was held in Cape Town on Thursday 30 June; but the Chairman and Vice-Chairman of Federal Council feel that there must be many other members of the Association who may wish to recognize the valuable services of Dr. Higgins by making a further presentation.

Any member who wishes to take part in this presentation should send his cheque, made out to the 'Dr. Shadick Higgins Presentation Fund', to the Secretary, M.A.S.A., P.O. Box 643, Cape Town.

## NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

### LAEVORAL

Westdene Products (Pty.) Ltd. announce the introduction of Laevoral, manufactured by Calmic, of England, and supply the following information:

Laevoral is a concentrated solution containing 65% of laevulose. It is particularly useful for long-term carbohydrate supportive therapy.

The extensive chemical and biochemical assessment of laevulose points to the following therapeutic advantage over glucose: It acts independently of insulin; it enters the carbohydrate metabolic system more directly; in conditions of severe liver damage the liver is capable of metabolizing it when it can no longer metabolize glucose; and its rapid clearance from the blood stream followed by its prompt conversion into glycogen gives a quick response in conditions of severe metabolic stress.

The use of Laevoral is suggested for hepatic conditions, for intoxications due to ingested or endogenous materials (hypnotic, alcoholic, uraemic, eclamptic, etc.) and for pre- and post-operative use. Laevoral may also be used as a sweetening and energizing agent in all conditions where domestic sugar is contra-indicated. In coeliac disease it is the sugar most readily assimilated, despite the existing hypersensitivity to sugars often exhibited in such cases.

Further details may be obtained from the sole South African distributors, Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

## TRESCATYL

Maybaker (S.A.) (Pty.) Ltd. announce the introduction of a new product Trescatyl brand ethionamide, and supply the following information:

Trescatyl is indicated for the treatment of pulmonary tuberculosis in cases where two or more of the standard antituberculosis drugs are unsuitable because of the presence of drug-resistant organisms or other contra-indications.

Where possible, sensitivity of the patient's organisms to available drugs and to ethionamide should be determined before treatment is started. Trescatyl is given orally in a dosage of 0·5 - 1 g. daily in 2, 3 or 4 portions and it must be administered in conjunction with the most suitable of the other available antituberculosis drugs.

Trescatyl is available in tablets of 250 mg. in containers of 250. Further information is obtainable from Maybaker (S.A.) (Pty.) Ltd., P.O. Box 1130, Port Elizabeth.

# BOOK REVIEWS : BOEKBESPREKINGS

## PUBLIC HEALTH IN BRITAIN

*Recent Advances in Public Health.* 2nd edition. By J. L. Burn, M.D., D.Hy., D.P.H. Pp. xi + 370. 54 illustrations. 40s. net. London: J. & A. Churchill Ltd. 1959.

The second edition of this book sets out, and very satisfactorily succeeds in covering, those aspects of the social and public-health facets which are particularly applicable to the present-day British welfare state. It adequately highlights the recent advances that have occurred since the printing of the first edition.

The author details the health and social facilities provided not only by the local authority, but also by the many voluntary welfare organizations which, even in the British welfare state, are accepted as also having an important part to play in augmenting the social and public-health services of that country. The chapter on health education, in particular, is most enlightening and can be accepted as a model of what we in this country will have to aim at. Other subjects that are ably discussed include the problem family and residential institutions for its rehabilitation, and air pollution, with its cost in ill health—particularly amongst the very young and the elderly—damage to buildings, and dislocation of transport services.

Dr. Burn's views on very early immunization against diphtheria, whooping cough and tetanus may not meet with full approval. Even today, there is great disagreement on the optimum time for starting the course.

This little treatise is admirably and clearly written, the paper is of excellent quality, and the list of references for further reading is fairly adequate. Perusal of its contents gives an excellent idea of the trend of the health and welfare services in the United Kingdom at the present time, and although we in this country have not reached the stage where all the social services listed and discussed could be provided here, we can nevertheless appreciate just what is being done to make the British people a healthy nation in the wider concept of that term.

I have no hesitation in recommending this book to all those who are anxious to know the manner and form of health and social services now available to the masses in the United Kingdom. We cannot yet in this country emulate these services; nevertheless there are many which could be provided in our large cities at small cost. Amongst these are the institutional care of the aged, where this is required, and the provision of clubs or a visiting roster to combat that extreme loneliness from which aged persons living alone so often suffer. This would be to the lasting benefit of a group who have shared in making this country what it is today, and have deserved well of their fellow men. E.D.C.

## CLINICAL PSYCHOPATHOLOGY

*Clinical Psychopathology.* By K. Schneider. Pp. xvi + 173. $4.50. New York and London: Grune & Stratton, Inc. 1959.

In translating Professor Schneider's second book, Miss Hamilton again has made accessible extremely stimulating views which no psychiatrist can afford to neglect. Many will be alienated by Schneider's underestimation of the significance of emotional experiences in the formation of (abnormal) personality, and his complementary insistence on an inborn constitutional basis for personality disorder, and his consequential critical view about the effectiveness of psychotherapy in treating personality disorders.

He does accept that external and internal stress can lead to mental reactions, but he does not consider such 'acute reactions to experience' to be illnesses. In his view, personality make-up plays a greater part in determining the form of the symptoms than the actual nature of the stressful experience. He tends to view psychoneurosis as a psychopathic reaction. In fact, he holds that the very concept of 'neurosis' is obsolete; no 'nerves' are disturbed according to modern psychopathology, and the patient requires to learn that he does not have a neurosis but is a neurotic—and to gain some insight into this is the first thing he has got to do. Schneider evidently rejects as mere conjecture much of the recent psychotherapeutic and sociological understanding of minor mental illness.

Psychopathic personality, concerning which he has made his major contribution to psychiatry, is likewise considered not an illness, but an abnormal variety of sane mental life. The section of the book which is most valuable and profound is the long chapter dealing with the phenomenology of the psychoses. Schneider holds the view that schizophrenia and manic-depressive psychosis are somatically caused, by an as yet undiscovered physical process. Diagnostic symptoms in schizophrenia depend on the loss of the contours of the self (loss of identity), and are thought-withdrawal, passivity thinking, diffusion of thought, and all passivity experiences. Delusional perception is also considered a diagnostic finding, and the discussion of this psychopathological phenomenon is masterly. In manic-depressive psychosis there are no such diagnostic symptoms, the morbid depression of mood being the nearest to a basic clinical finding. However, while schizophrenia has such characteristic and vivid symptomatology, manic-depressive psychosis has become far more clearly demarcated and prognostically the most certain of the endogenous psychoses. H.W.

## HISTOLOGY

*Histologie und mikroskopische Anatomie des Menschen.* By Dr. Wolfgang Bargmann. Pp. xv + 820. 658 illustrations. DM 69.60. Stuttgart: Georg Thieme Verlag. 1959.

This is the 3rd edition of this book within 8 years. What a change in comparison with the 1st edition!

Bargmann deals firstly with the cell, then with the tissues, and lastly with the different organs. The general part of this book has been considerably revised. Electron-microscope findings and histochemical results are integrated with those of ordinary microscopy. For this reason the histological sections of this book are excellent and present the student with a very good introduction in this difficult science. Bargmann explains the microscopical anatomy, and here one cannot criticize the length of a particular chapter or the lack of detail in another, for these considerations depend upon the interests and intentions of the author. All the sections of this book are adequate for use by students. The illustrations, mostly black-and-white, are good, and the general lay-out demonstrates the author's experience and the publisher's craftmanship. H.W.W.

## STEAM STERILIZATION

*The Operation of Sterilising Autoclaves.* A Symposium held at Brighton Technical College, School of Pharmacy, on 9 May 1959. Pp. 45. Illustrations. 7s. 6d. + 4d. postage. Obtainable from A.P.S. Journal Pty. Ltd., P.O. Box 6290, Johannesburg. London: The Pharmaceutical Press. 1959.

This report is clear, concise, and enlightening. Bearing in mind the recent realization of inadequate and defective sterilization procedure in hospitals, the articles are of special benefit to superintendents, and even to laymen in charge of hospitals.

The report deals briefly with the principles underlying steam sterilization. The chapter on the design of pressure steam sterilizers is rather technical, but it gives a clear lead about the features of an efficient steam sterilizer. The part dealing with methods of

testing the bactericidal efficiency of steam sterilizers gives useful guidance. The best section is certainly that on the 'operation and use of sterilizing equipment and staff responsibilities'.

With the grave danger of cross-infection in hospitals constantly facing the hospital administrator, the subjects dealt with, although not new, will certainly aid in bringing about a better understanding and handling of the problem. J.dB.

## TRANSPLANTABLE AND TRANSMISSIBLE TUMOURS

*Atlas of Tumour Pathology - Section XII - Fascicle 40 - Transplantable and Transmissible Tumours of Animals.* By H. L. Stewart, M.D.; K. C. Snell, M.D.; L. J. Dunham, M.D. and S. M. Schlyen, M.D. Pp. 378. 287 figures. $3.50. Washington: Armed Forces Institute of Pathology. 1959.

This fascicle matches in all details the high standard of its predecessors but is unlike them in not attempting a complete survey of a particular field of tumour pathology. The authors have instead tried 'to select representative examples of the great variety of transplantable tumours that commonly occur spontaneously or that can be induced in different animal species'. Fifty tumours from a wide variety of sites are described; they include tumours found in mice, rats, rabbits, dogs, fowls, hamsters, guinea-pigs and frogs. Tumours were chosen for their commonness, for their historical significance, or to illustrate the effects of hormones and endocrine glands. Each tumour is given the same treatment; this includes the history of the spontaneous disease or original tumour, transmission or transplantation studies, a description of the gross appearances, and a very detailed account of the microscopic appearances. In some cases the bearing of a particular tumour on cancer research in general and current theories of carcinogenesis is briefly mentioned; the only serious criticism one can offer on this fascicle is that the authors appear to have been prevented, presumably by space, from making sufficient comment of this more general type. J.A.H.C.

## BREAST CANCER

*Cancer of the Breast.* Compiled and edited by Willard H. Parsons, M.D., F.A.C.S. Pp. xv + 232. Figures. 60s. Oxford: Blackwell Scientific Publications Ltd. 1959.

This is one of the most lucid, straightforward and honest monographs your reviewer has ever had the good fortune to come across. The information given is accurate, the literature cited extensive, and the judgments balanced and massive. The format lends itself to easy reading and the illustrations do indeed illustrate.

It may be invidious to select certain chapters for special praise, but F. W. Foote's chapter on pathology and J. A. Urban's on the supra-radical operations are quite outstanding. On the other hand, the chapter on hormone treatment, as it does less than full justice to this important subject, fails to impress by comparison with the others.

One can thoroughly recommend this book to the practising surgeon, the surgical teacher and the postgraduate student. It sums up most of the facts that are known today about cancer of the breast and would be a help to anyone in making up his mind what to do in any eventuality. It was particularly pleasant not to find still another classification put forward, and the chapter on pre- and post-operative management has a far wider application than merely to the subject mentioned in the title of the book. T.S.

## INFANT FEEDING

*Infant Feeding and Feeding Difficulties.* 3rd edition. By P. R. Evans, M.D., M.Sc., F.R.C.P. and Ronald MacKeith, M.A., D.M., F.R.C.P., D.C.H. Pp. viii + 293. 66 illustrations, including 2 coloured plates. 16s. net. London: J. & A. Churchill Ltd. 1958.

This useful book, now in its 3rd edition, should be useful to the large group of people, medical and non-medical, who are concerned with the management of infant feeding and feeding problems.

The introductory chapters on the physiology of lactation, the infant's digestive system and the nutritive needs of the infant and its mother, are particularly well done. Breast feeding is also adequately dealt with. As in all books on infant feeding, there are likely to be controversial views about any particular approach to artificial feeding. The use of cow's milk is well described, but in countries such as South Africa, in which dried milk is extensively used, readers would have liked more information and illustrative formulae on dried milk. Hardly anything is said about the time or method of introducing solids, and attention should certainly be paid to this important aspect of infant feeding in future editions.

All-in-all this book can be safely recommended to anyone interested in modern methods of infant feeding. I.M.

## DISINFECTION

*Disinfection and Sterilization.* By G. Sykes, M.Sc. (Lond.), F.R.I.C. Pp. xviii + 396. 17 figures. 7 plates. 75s. net. London: E. & F. N. Spon Ltd. 1958.

This publication represents a thorough and up-to-date account of the subjects of sterilization and disinfection. Theoretical aspects of the subjects are dealt with in great detail without neglecting the practical side, such as methods of testing disinfectants and the various methods employed in sterilization. A section devoted to the disinfection of viruses gives useful information which was previously lacking in older books.

This is a useful reference book which should prove invaluable to bacteriologists, public-health officials, hospital personnel, and others concerned with large-scale sterilization procedures.

The book is written in a clear style and the wide field it covers is well indexed both for authors and subjects. W.dT.N.

## YEAR BOOK OF OBSTETRICS AND GYNAECOLOGY

*Year Book of Obstetrics and Gynaecology, 1959-1960.* Edited by J. P. Greenhill, B.S., M.D., F.A.C.S., F.I.C.S. (Hon.). Pp. 573. $8.00. Chicago: The Year Book Publishers. 1959.

The 1959-60 Year Book follows the pattern of previous editions, giving an indexed summary of the literature for the year ending September 1959, with useful annotations by the editor. Apart from a few references to Dutch, German and South American publications, only articles in the English language are covered.

As with previous publications, the obstetrical subject matter is grouped under the headings of physiology, abortion, ectopic pregnancy, complications, the toxaemias, labour, analgesia and anaesthesia, complications of labour, operative obstetrics, haemorrhage, puerperium, and the newborn. In the gynaecology section the headings are: General principles and diagnosis, infertility, operative gynaecology, infections, malignant tumours, menstrual disorders, endocrinology, report of conference on the uterus held by the New York Academy of Sciences, and extracts from presidential addresses.

This book certainly lessens the task of keeping abreast with the multitude of interesting publications found today and as such can be recommended. J.N.dV.

## PHYSIOLOGY IN SURGERY

*Pathophysiology in Surgery.* By James D. Hardy, M.S. (Chem.), M.D., F.A.C.S. Pp. xx + 704. 278 figures. 152s. London: Baillière, Tindall and Cox Ltd. 1958.

This volume by a single author is an attempt to cover problems of surgical physiology. The subject matter, however, is broader than this and in parts provides comprehensive discussion of types of surgical operations and complications.

The opening chapters, on the physiology of injuries, fluid balance, nutrition, and wound healing, are excellent. They are informative and well written and provide detailed postgraduate reading. From then on the volume becomes erratic, with poor chapters on burns, an incomplete review of renal physiology and pathology, and a sketchy description of head injuries. There is a surprising lack of discussion of any physiology of bone or cartilage.

The chapters on circulation and the alimentary tract occupy a considerable portion of the book and much of each chapter is devoted to unnecessary surgical detail. To those with knowledge and training in British and Continental techniques the book is insular, being confined largely to American contributions.

The title *Pathophysiology in Surgery* is an unwieldy one: but this book is one of a series of books published recently on basic problems confronting the surgeon and, as such, it is a contribution in stimulating surgical thinking along these lines.

If it were a better balanced book and if all chapters were as penetrating and interesting as the first 4 it would be essential for postgraduate reading. As it is, the book provides an erratic

coverage of the detailed aspects of surgical physiology, some excellent, some poor. The references are selective, whilst the publication itself is well produced, with informative illustrations.
A.E.K.

## ALCOHOL AND MOTOR DRIVING

*Effect of Small Doses of Alcohol on a Skill Resembling Driving.* By G. C. Drew, M.A.; W. P. Colquhoun, M.A., Ph.D. and Hazel A. Long, B.Sc. Pp. vi + 108. Illustrations. 7s. 6d. net. London: Her Majesty's Stationery Office. 1959.

The authors, using the ingenious 'Miles motor driving trainer', studied basic skill similar to driving in 40 volunteers.

On each of 5 2-hourly sessions, subjects received one of 4 alcohol doses—resulting in blood-alcohol concentrations of roughly 20, 40, 60 and 80 mg.%.—or a placebo dose.

It was demonstrated conclusively that performance began to deteriorate with blood alcohol levels of 20 - 30 mg.% and that the deterioration, in general, progressed as the blood-alcohol level rose.

The results were evaluated strictly in relationship to the object of the study—a difficult task, which could have been approached in no other or better fashion. For this reason it would be dangerous to assume that experimental performances are indicative of driving skill and judgment on the actual road. However, the existence of a 'sub-intoxicated' state is proved beyond any question, and should be accepted by our legislators if the mounting toll on our roads is to be checked.
C.K.E.

## CLINICAL CHEMICAL PATHOLOGY

*Clinical Chemical Pathology.* 2nd edition. By C. H. Gray, M.R.C.P., F.R.I.C. Pp. vii + 160. Illustrations. 14s. net. London: Edward Arnold (Publishers) Ltd. 1959.

The present edition of this excellent text-book for students has been revised in part, and where knowledge has become more complete the current views have been incorporated in the present text. Thus the relationship between the glucuronide conjugates of bilirubin and free bilirubin to the Van den Berg reaction is discussed under Liver Function Tests; potassium deficiency is dealt with more adequately; and the chapter on the Chemical Pathology of the Alimentary Tract has been curtailed and tests considered to be outmoded eliminated from the appropriate texts. The section on Endocrines includes some of the more recent advances in this field and there is the welcome addition of transaminase estimations and a mention of some of the rare metabolic disorders.

The chapter on Fats has been rewritten completely and this is the only change which disappoints. Brevity in this instance has been pushed to the point where the student may well be confused. However, this criticism in no way detracts from the general excellence of this book, and there can be little doubt that not only students but registrars and practitioners in general will continue to consult this most concise, clearly written book on Chemical Pathology.
S.M.J.

## PSYCHOPATHY

*Psychopathy.* By C. Frankenstein, Ph.D. Pp. viii + 198. $6.75. New York and London: Grune & Stratton, Inc. 1959.

It has long been recognized that the concept 'psychopath' is as inadequate as the elusive character it purports to describe. This book is an attempt to define psychopathy in restrictive terms which exclude other conditions related to it through mere symptomatic similarity. Neurotic character disorders, perversions and antisocial behaviour patterns as such, are discarded by the author as failing to conform to his selective basic criteria on which alone he permits a diagnosis of psychopathy to be reached. He maintains that 'certain structural dispositions' present at birth or shortly after 'determine the development and crystallization of the final psychopathic reaction types'. These results of 'structurization' are irreversible.

The therapeutic pessimism implicit throughout this treatise makes the reader sometimes wonder if the semantic obstacle race is worth the trouble in the end, for the going is much heavier than with Cleckley's masterly monograph, and the conclusions get us no further.
A.M.L.

## ARTERIAL EMBOLISM

*Arterial Embolism in the Limbs. The Clinical Problem and its Anatomical Basis.* By A. L. Jacobs, M.A., D.M. (Oxon), F.R.C.P. Pp. xii + 200. Illustrations. 35s. net + 1s. 10d. postage abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1959.

This interesting monograph on limb embolism is based on the study and analysis of widely assorted clinical experience and morbid-anatomy material gathered by Dr. Jacobs over a period of 16 years. He describes the diagnosis and assesses the value of various methods of treatment and finally summarizes his main conclusions. This valuable discussion is obviously the result of long and painstaking clinical observation of a large number of patients, and the eventual outcome of their treatment or non-treatment.

Dr. Jacobs discusses, among other facets, the value of embolectomy and, although the relatively small number of successes is disappointing, he emphasizes that this is often attributable to contributory factors (e.g. tardy diagnosis) and he stresses the necessity for individual consideration of cases for operation because embolectomy still takes a first place in the treatment of embolism of the larger limb arteries.

Although arterial embolism is a condition fairly frequently met with in general practice, there is little comprehensive literature available on the subject, and this clear and complete survey, illustrated with many cases histories, tables for comparison, and X-ray plates, will be well appreciated by the practitioner.
C.N.B.

## BOOKS RECEIVED : BOEKE ONTVANG

*Appraisal of Fellowships.* Report of a Study Group. Technical Report Series No. 186. Pp. 15. 1s. 9d. Also available in French and Spanish. Geneva: World Health Organization. 1960. Local agent: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria.

*Expert Committee on Addiction-producing Drugs.* Tenth Report. Technical Report Series No. 188. Pp. 16. 1s. 9d. Also published in French and Spanish. Geneva: World Health Organization. 1960. Local agent: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria.

*Expert Committee on Hygiene and Sanitation in Aviation.* 1st report. Technical Report Series No. 174. Pp. 62. 3s. 6d. Also published in French and Spanish. Geneva: World Health Organization. 1959. Local Sales Agent: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria.

*A Text-book of X-ray Diagnosis.* Vol. 4. 3rd edition. Edited by S. Cochrane Shanks, *C.B.E.*, M.D., F.R.C.P., F.F.R. and Peter Kerley, *C.V.O., C.B.E.*, M.D., F.R.C.P., F.F.R., D.M.R.E. Pp. xvi+714. 735 illustrations. London: H. K. Lewis & Co. Ltd. 1959.

*Color Atlas and Management of Vascular Disease.* By William T. Foley, M.D., F.A.C.P. and Irving S. Wright, M.D., F.A.C.P. Pp. xi+170. 194 illustrations. $18.00. New York: Appleton-Century-Crofts, Inc. 1959.

*Surgical Service Guide.* By Louis T. Palumbo, M.D., M.S., F.A.C.S. Pp. 208. Illustrations. $6.00. Chicago: Year Book Publishers, Inc. 1959.

*The Year Book of Cancer* 1958-59. Compiled and edited by R. L. Clark, Jr., B.S., M.D., M.Sc. (Surgery), D.Sc. (Hon.) and R. W. Cumley, B.A., M.A., Ph.D. Pp. 570. 202 figures. $8.00. Chicago: Year Book Publishers, Inc. 1959.

*The Calendar for the Modern Age.* By Elisabeth Achelis. Pp. 220. $4.00. Edinburgh, New York, Toronto: Thomas Nelson & Sons. 1959.

*Aspecten van Arbeid en Arbeidstherapie in de Psychiatrische Inrichting.* By H. van der Drift. Pp. 135. f 5.90 ing. Arnhem: Van Loghum Slaterus. 1959.

*L'Hypnose, Problèmes Théoriques et Pratiques.* By. L. Chertok. Pp. 80. 700 fr. Paris: Masson et Cie. 1959.

## CORRESPONDENCE : BRIEWERUBRIEK

### RADIO-ACTIVE ISOTOPES AND FEES TO BE CHARGED BY SPECIALIST PHYSICIANS FOR ADMINISTERING THERAPEUTIC DOSES

*To the Editor:* In the report[1] of the 71st meeting of the South African Medical and Dental Council, which was published in the *Journal* for 14 May, it is stated (under the section 'Radio-active isotopes') that 'No. 13 . . . of the ethical rules does not preclude a specialist physician from administering radio-active isotopes therapeutically, provided he does not make an extra charge for the procedure . . .'. It is also stated (correctly) that 'whether the Atomic Energy Board would be prepared to authorize him to use radio-active isotopes is not a matter within the purview of the Council'.

This does not altogether clarify the present position. The only practitioners permitted to administer therapeutic doses of radio-active isotopes are radiologists and these, apart from holding the Diploma in Radiotherapy or Radiology, must have special authority from the Atomic Energy Board through the Radio-active Isotope Committee to use radio-active isotopes. In other words, it is not even every radiotherapist who is permitted to administer therapeutic quantities of radio-active isotopes. The radiotherapist must satisfy the Atomic Energy Board that he has had special experience in the subject and that his premises have been inspected by the physicist of Atomic Energy Board and found satisfactory.

No consultant or specialist other than radiotherapists or radiologists has so far been given authority to administer therapeutic doses of radio-active isotopes. The question of what fees physicians are to charge, therefore, does not arise.

The following is the gist of the rules laid down by the Atomic Energy Board for the administration of the therapeutic doses of radio-active isotopes (A doses).

1. The practitioner must be a qualified radiotherapist or radiologist recognized by the Atomic Energy Board as a radiotherapist qualified to use radio-active isotopes.

2. Physicians and research workers may use B doses for diagnostic purposes provided they have obtained authority from the Atomic Energy Board, and this authority would be based on an opinion of the Medical Isotope Committee, that the applicant has had sufficient experience in the use of radio-active isotopes and that his premises or laboratory have been found satisfactory by the Atomic Energy Board physicist.

It must be noted, too, that permission to use radio-active isotopes is not granted automatically even to radiotherapists and, if they cannot supply proof that they have had the necessary experience, the Atomic Energy Board may arrange an examination for radiotherapists, as in fact, it did at the beginning of this year, following a course organized by Dr. J. Muir Grieve at Groote Schuur Hospital, Cape Town.

M. Weinbren.
X-ray Department
Chamber of Mines Hospital
P.O. Box 774
Johannesburg
30 June 1960

1. South African Medical and Dental Council, Report (1960): S. Afr. Med. J., 34, 405.

### RECOVERY ROOMS

*To the Editor:* My attention has been drawn to item 111 of the Minutes of the Meeting of the Federal Council of the Medical Association of South Africa held on 3 - 5 March 1960, as reported in the issue of the *Journal*[1] dated 4 June 1960. This refers to the resolution of the South African Society of Anaesthetists which strongly urges all hospitals, nursing homes and similar institutions to provide recovery rooms staffed by suitably trained registered nurses.

In the new Johannesburg Nursing Home By-laws now with the Administrator for promulgation, provision is in fact made for a recovery room in every operating unit with, however, the proviso that the recovery room need not be provided in respect of an existing nursing home where the Medical Officer of Health is satisfied that it cannot be complied with at reasonable expense or without undue prejudice to the existing facilities for operating units.

J. W. Scott Millar
*Medical Officer of Health*
City of Johannesburg
City Health Department
P.O. Box 1477
Johannesburg
27 June 1960

1. Minutes of Meeting of Federal Council held in Pretoria on 3, 4 and 5 March 1960 (1960): S. Afr. Med. J., 34, 484.

### PLAQUE IN MEMORY OF THE LATE MR. T. LINDSAY SANDES

*To the Editor:* The medical staff of the Victoria Hospital, Wynberg are delighted with the acquiescence of the Provincial Administration to the suggestion that the new European Block at this hospital be named after the late Mr. T. Lindsay Sandes, who for so many years added lustre to the fame of the hospital by his presence on its honorary staff.

It is proposed to erect a plaque in the entrance to this building in memory of Mr. Lindsay Sandes. The cost could easily be met in part or altogether by contributions from the staff. To date, this represents the only memorial to this outstanding citizen and member of the medical profession. The staff of the hospital are of the opinion that there is a considerable number of people, both lay and medical, who would like to participate in such a memorial.

For this reason, it is suggested that contributions from interested persons should be sent to the Secretary of the Medical Staff, Victoria Hospital, Wynberg. These would be duly acknowledged and a donors list prepared. The plaque is by no means expensive and this proposal is aimed at giving the widest possible body of people an opportunity of contributing to the memorial.

J. Silberbauer
*Hon. Secretary, Medical Committee*
Victoria Hospital
Alphen Hill
Wynberg, Cape
28 June 1960

### TENTH INTERNATIONAL CONGRESS OF RADIOLOGY

*To the Editor:* My Society would appreciate it if you would publish the following information in the *Journal,* as soon as possible:

The Tenth International Congress of Radiology will be held from 26 August to 1 September 1962 in Montreal, Canada.

The Radiological Society of South Africa has been requested to send a delegation consisting of a Chairman and 5 alternates, one of whom should be designated as the alternate chairman.

Any radiologist who wishes to attend this Congress as an official member of the South African delegation, is requested to communicate with the undersigned, not later than 15 January 1961.

Cecil Komins
*Hon. Secretary*
Radiological Society of
South Africa (M.A.S.A.)
1 Lister Buildings
Jeppe Street
Johannesburg
29 June 1960

### NAUDÉ APPEAL FUND

*To the Editor:* I list hereunder further donations received for the Naudé Appeal Fund and would appreciate it if you would kindly publish the list in the *Journal:*

An amount of £7 7s. 0d. has been received from Drs. W. Gilbert, Hendrik Muller and Prof. J. F. Brock, of Cape Town, and Dr. J. P. Kruger, of Johannesburg.

J. J. Hofmeyr
*Hon. Secretary*
Transkei Branch (M.A.S.A.)
P.O. Box 318
Umtata
20 June 1960